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Parental bonding, attachment, reality discrimination, and psychotic-like experiences

David Smailes, B.Sc., M.Sc.

A thesis submitted for the degree of Doctor of Philosophy in the Department of Psychology
at Durham University

December 2013

Table of Contents

List of Tables.....	iv
List of Figures.....	v
Declaration.....	vi
Statement of Copyright.....	vii
Acknowledgements.....	viii
Thesis Abstract.....	ix
Publications included in this thesis.....	xii
 Chapter 1. General Introduction.....	 1
1.1 Introduction.....	1
1.2 Parental bonding and psychotic experiences.....	5
1.3 Attachment style and psychotic experiences.....	10
1.4 Reality discrimination and auditory hallucinations.....	15
1.5 Socio-economic factors and psychotic experiences.....	17
1.6 Chapter summary and outline of thesis.....	18
 Chapter 2. Study 1: Associations between parental bonding, negative affect, bullying, and psychotic-like experiences in a non-clinical sample of adolescents.....	 21
2.1 Abstract.....	22
2.2 Introduction.....	23
2.3 Method.....	25
2.4 Results.....	28
2.5 Discussion.....	36

Chapter 3. Study 2: Associations between attachment style, loneliness, and psychotic-like experiences in a non-clinical sample of adults.....	40
3.1 Abstract.....	41
3.2 Introduction.....	42
3.3 Method.....	43
3.4 Results.....	46
3.5 Discussion.....	52
Chapter 4. Study 3: Associations between attachment style, anomalous percepts, and paranoid thinking in a non-clinical sample of adults.....	56
4.1 Abstract.....	57
4.2 Introduction.....	58
4.3 Method.....	61
4.4 Results.....	63
4.5 Discussion.....	69
Chapter 5. Study 4: Associations between intrusive thoughts, reality discrimination, and auditory hallucination-proneness in a non-clinical sample of adults.....	73
5.1 Abstract.....	74
5.2 Introduction.....	75
5.3 Method.....	77
5.4 Results.....	80
5.5 Discussion.....	87

Chapter 6. Study 5: The impact of negative affect on reality discrimination.....	92
6.1 Study 5 Pilot.....	88
6.2 Abstract.....	96
6.3 Introduction.....	97
6.4 Method.....	100
6.5 Results.....	104
6.6 Discussion.....	107
 Chapter 7. General Discussion.....	 111
7.1 Summary of findings.....	111
7.2 Theoretical implications.....	115
7.3 Directions for future research.....	118
7.4 Limitations.....	124
7.5 Summary.....	130
 References.....	 131

List of Tables

Table 2.1 Descriptive statistics for, and correlations between, measures of parental bonding, bullying, affect, hallucination-proneness, and paranoid thinking.....	29
Table 2.2 Summary of hierarchical regression analysis for hallucination-proneness.....	31
Table 2.3 Summary of hierarchical regression analysis for paranoid thinking.....	32
Table 2.4 Mediation of the association between maternal care and PLEs by bullying and negative affect.....	34
Table 3.1 Descriptive statistics for, and correlations between, measures of attachment, loneliness, negative affect, hallucination-proneness, and paranoid thinking.....	48
Table 3.2 Summary of hierarchical regression analysis for paranoid thinking.....	50
Table 3.3 Mediation of the association between attachment style and paranoid thinking by loneliness.....	52
Table 4.1 Descriptive statistics for, and correlations between, measures of attachment, anomalous percepts, negative affect, and paranoid thinking.....	65
Table 4.2 Summary of hierarchical regression analysis for paranoid thinking.....	66
Table 5.1 Descriptive statistics for, and correlations between, measures of negative affect, intrusive thoughts, number of false alarms, and auditory hallucination-proneness.....	82
Table 5.2 Summary of hierarchical regression analysis for auditory hallucination-proneness.....	84
Table 6.1 Descriptive statistics for jumbled speech task performance.....	94
Table 6.2 Descriptive statistics for all study variables.....	105

List of Figures

Figure 4.1 Moderation of the association between anomalous percepts and paranoid thinking by attachment anxiety.....	68
Figure 5.1 Interaction between level of intrusive thoughts and number of false alarms made during a reality discrimination task.....	85
Figure 5.1 Interaction between level of intrusive thoughts and β (response bias) during a reality discrimination task.....	87
Figure 6.1 Mean (a) number of false alarms, (b), number of hits, (c), d' and (d) β in the three mood induction groups.....	107
Figure 7.1 A model of AH consistent with the data presented in this thesis.....	119
Figure 7.2 A model of paranoid thinking consistent with the data presented in this thesis.....	120

Declaration

I, David Smailes, confirm that the work presented in this thesis is my own. Where information has been derived from other sources, I confirm that this has been indicated in the thesis.

This thesis was prepared in accordance with the guidelines outlined by Durham University's Graduate School and in the Department of Psychology's Postgraduate Handbook. References have been amalgamated at the end of the thesis for ease of reading.

A handwritten signature in black ink that reads "D. Smailes". The script is cursive and fluid, with the first letter 'D' being large and prominent.

David Smailes

Statement of Copyright

The copyright of this thesis rests with the author. No quotation from it should be published without the author's prior written consent and information derived from it should be acknowledged.

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Thesis Abstract

Psychological models of psychotic experiences (e.g., Bentall & Fernyhough, 2008; Freeman, 2007; Waters et al., 2012) suggest that social adversity (e.g., difficult family relationships, bullying) and anomalous percepts play an important role in the development of paranoid thinking, while problems in reality discrimination and intrusive cognitions play an important role in the development of auditory hallucinations (AH). The studies reported in this thesis examined a number of research questions relevant to these models. Across these studies, it is assumed that investigating psychotic experiences in non-clinical populations (typically referred to as psychotic-like experiences, or PLEs; Kelleher & Cannon, 2011) is valuable in understanding the development of clinical psychotic experiences.

The first group of studies (Studies 1, 2, and 3) examined the ways in which poor parental bonding (low perceived care, high perceived overprotection) or insecure peer attachment relate to PLEs. In Study 1, associations between parental bonding, bullying, negative affect, paranoid thinking, and AH-proneness were examined in a non-clinical sample of adolescents. It was predicted that bullying and negative affect would mediate the association between parental bonding and PLEs. In line with this prediction, the associations between maternal care and paranoid thinking and between maternal care and AH-proneness were mediated by bullying and negative affect. None of the other aspects of parental bonding (i.e., maternal overprotection, paternal care, and paternal overprotection) were independent predictors of paranoid thinking or AH-proneness.

In Study 2, associations between peer attachment style, loneliness, and PLEs were examined in a non-clinical sample of young adults, who completed an online questionnaire pack. It was predicted that loneliness would mediate the association between attachment style and PLEs. Consistent with this prediction, loneliness mediated the association between

attachment style and paranoid thinking. In contrast, neither attachment style nor loneliness were associated with AH-proneness. In Study 3, the associations between anomalous percepts, attachment style, and paranoid thinking were investigated in a non-clinical sample of young adults. It was predicted that attachment anxiety would moderate the association between anomalous percepts and paranoid thinking. A moderating effect of attachment anxiety was found, but it was different to the predicted interaction effect. When participants experienced relatively infrequent anomalous percepts, individual differences in attachment anxiety were important in predicting which participants were likely to report low levels of paranoid thinking, and which participants were likely to report high levels of paranoid thinking. However, when participants experienced frequent anomalous percepts, individual differences in attachment anxiety were not important in predicting which participants were likely to report high levels of paranoid thinking. Thus, the findings of Studies 1, 2, and 3 show how a variety of social factors (e.g., attachment style and maternal care) interact in complex ways with other social (e.g., bullying), emotional (e.g., negative affect), and cognitive (e.g., anomalous percepts) factors to foster or preclude the development of PLEs in ways that extend current psychological models of AH (Bentall & Fernyhough, 2008) and paranoid thinking (Freeman, 2007).

The second group of studies examined (a) the role reality discrimination plays in non-clinical hallucinatory experiences, and (b) whether reality discrimination can be modulated in non-clinical samples of young adults by manipulating their mood. In Study 4, associations between reality discrimination biases, intrusive thoughts, and AH-proneness were examined in a non-clinical sample of young adults. It was predicted that the association between intrusive thoughts and AH-proneness would be moderated by biased reality discrimination. Consistent with this prediction, participants who made a large number of reality discrimination errors and reported high levels of intrusive thoughts were especially prone to

hallucinatory experiences. In Study 5, the effects of negative emotion on participants' reality discrimination abilities were examined. Participants who received either a loneliness induction or a negative affect induction showed biased reality discrimination, in comparison to control participants. That is, participants who completed either of the two negative mood inductions made more external misattributions than did participants who completed the neutral mood induction. These findings suggest that mood modulates reality discrimination, and it is argued that this may help to account for the episodic nature of AH. Thus, the findings of Studies 4 and 5 show how cognitive factors (e.g., reality discrimination abilities and intrusive thoughts) interact with one another, and with emotional factors (e.g., negative affect), to foster or preclude the development of AH in ways that extend current psychological models of AH (Bentall & Fernyhough, 2008, Waters et al., 2012).

Together these five studies advance our understanding of (a) the roles played by parental bonding and attachment style in the development of PLEs, (b) the potential role played by reality discrimination in the development of hallucinatory experiences, and (c) how reality discrimination abilities can be modulated by mood. The implications of these findings for cognitive-developmental models of psychotic experiences, as well as directions for future research, are discussed in the General Discussion.

Publications included in this thesis

Two of the studies reported here have been submitted for publication and were under review at the time that this thesis was submitted for examination. A manuscript based on the study reported in Chapter 5—*Associations between intrusive thoughts, reality discrimination, and auditory hallucination-proneness in a non-clinical sample of adults*—is under review at *Cognitive Neuropsychiatry*. A manuscript based on the study reported in Chapter 6—*The impact of negative affect on reality discrimination*—is under review at *Journal of Behavior Therapy and Experimental Psychiatry*.

Chapter 1

General Introduction

1.1 Introduction

The term *psychosis* refers to a loss of contact with reality (Geyer & Vollenweider, 2008). Traditionally, psychotic experiences have been viewed as a symptom of schizophrenia or of a related disorder (e.g., schizoaffective disorder; American Psychiatric Association, 2013), although they are also often reported by patients diagnosed with other types of mental health problems (e.g., post-traumatic stress disorder; Butler, Mueser, Sprock, & Braff, 1996; borderline personality disorder; Slotema et al., 2012). Moreover, over the past 15 years, a large body of evidence has accumulated that shows that a substantial minority of the general population report psychosis-like experiences (Linscott & van Os, 2013).

Two of the most common forms of psychotic experiences are auditory hallucinations (AH) and paranoid thinking. AH refer to an auditory experience, perceived in the absence of an appropriate external stimulus, which “has a sufficient sense of reality to resemble a veridical perception, over which the subject does not feel s/he has direct and voluntary control, and which occurs in the awake state” (David, 2004, p. 110). AH are reported by 50% to 70% of patients diagnosed with schizophrenia (Andreasen & Flaum, 1991; Sartorius, Shapiro, & Jablensky, 1974). Persecutory delusions are beliefs held by a person that are not shared by others, are implausible, are resistant to change, are distressing, and that refer to harm that is occurring, or is going to occur, to him/her as a result of the intentions of a persecutor (Freeman & Garety, 2000). When a person reports persecutory beliefs that are not implausible (e.g., “People would harm me if they had the opportunity to do so” rather than “The government is involved in a conspiracy to murder me”) this is referred to as paranoid

thinking (Freeman & Garety, 2006). Persecutory delusions or paranoid thinking are reported by around 50% of people diagnosed with schizophrenia (Sartorius et al., 1986).

Other, less common, psychotic experiences include thought disorder, thought insertion, and other forms of delusional beliefs. Around 38% of patients diagnosed with schizophrenia experience thought disorder (Howard, Castle, Wessley, & Murray, 1993), which is defined as poverty of speech, incoherent speech, use of neologisms, and/or a loosening of the associations between ideas (Andreasen, 1986). Around 15% of patients diagnosed with schizophrenia report thought insertion (Howard et al., 1993; Sartorius et al., 1986), where a person believes that they are experiencing the thoughts of another person (Mullins & Spence, 2003). Up to 30% of patients diagnosed with schizophrenia report delusional beliefs which are not persecutory in nature (Howard et al., 1993; Sartorius et al., 1986). These include ideas of reference (e.g., where a person believes that neutral events, such as conversations in television programmes or newspaper articles, are referring to them, or to events in their life; Wong et al., 2012) and delusions of control (e.g., where a person believes that others can control their behaviour; Frith, Blakemore, & Wolpert, 2000).

Traditionally, bio-genetic models of psychosis (e.g., Weinberger, 1987) have dominated the field (Read, Mosher, & Bentall, 2004). Bio-genetic models typically propose that some form of biological abnormality is the causal factor driving the development of psychosis. For example, in Weinberger's (1987; Marengo & Weinberger, 2000) neurodevelopmental model, it is argued that some form of insult to the brain either *in utero*, at birth, or in the first few years of life, places a person at risk of developing psychosis. This brain insult may be a result of genetic factors (Harrison & Weinberger, 2005), maternal infection during pregnancy (Brown, 2006), hypoxia during labour (Geddes et al., 1999), or other adverse biological events, such as exposure to central nervous system infections (Rantakallio, Jones, Moring, & von Wendt, 1997). This brain insult is proposed to subtly

affect behaviour throughout childhood, but typically leads to the development of psychotic experiences during adolescence or adulthood following further neurobiological changes (e.g., Cannon et al., 2003). This claim is supported by studies that have reported subtle cognitive and behavioural alterations in young children who will be diagnosed with a psychotic disorder in adulthood. For example, Jones, Murray, Rogers, and Marmot (1994) reported that children who were later diagnosed with schizophrenia began walking at a later age than did children who were not diagnosed with schizophrenia in adulthood. Similarly, Cannon et al. (2002) reported that children who were later diagnosed with a schizophreniform disorder showed motor impairments, had lower IQ scores, and displayed lower receptive language skills in comparison to children who were not diagnosed with schizophrenia in adulthood.

Bio-genetic models like Weinberger's are, however, limited in a number of ways. For example, Bentall, Fernyhough, Morrison, Lewis, and Corcoran (2007) have argued that these models fail to account for (a) the age of onset of psychosis, (b) the role of the social environment in the development of psychotic experiences, and (c) the psychological abnormalities associated with specific symptoms. For example, in terms of the age of onset of psychosis, Bentall et al. (2007) argue that these models fail to provide a good account of why the peak age of onset of psychosis is in a person's mid-20s, rather than at any other age. Meanwhile, in terms of the role of the social environment in the development of psychotic experiences, bio-genetic models make only passing reference to the importance of social adversity, despite strong evidence that social adversity is associated with a large increase in the risk of developing psychotic experiences (Varese et al., 2012). Typically bio-genetic models identify social factors as variables that can, only in the presence of a biological vulnerability, trigger the onset of psychotic experiences (e.g., Tsuang, 2000). Finally, in terms of neurodevelopmental models failing to account for the psychological abnormalities associated with specific symptoms, Bentall et al. note that researchers have demonstrated that

different psychotic experiences are associated with specific cognitive biases, and suggest that this cannot be accounted for by a neurodevelopmental account. For example, psychotic participants who report delusional beliefs, but not those without delusional beliefs, show a jumping-to-conclusions bias, where they make decisions based upon very small amounts of information (Fine, Gardner, Craigie, & Gold, 2007). Meanwhile, as discussed in more detail below, psychotic participants who report AH, but not those without AH, show biased reality discrimination, where they tend to misattribute self-generated responses to a non-self source (Brookwell, Bentall, & Varese, 2013). Bentall et al. argue that neurodevelopmental models of psychosis struggle to explain how a single, static lesion could lead to different cognitive biases, which elicit different psychotic experiences.

It has been suggested that approaches that view psychotic experiences as endpoints of atypical social, emotional, and cognitive trajectories, can address these limitations (e.g., Bentall & Fernyhough, 2008; Bentall et al., 2007; Read, Perry, Moskowitz, & Connolly, 2001). For example, cognitive-developmental models are able to account for the age of onset of psychosis by explaining psychotic experiences as distressing variants of relatively normal phenomena in adolescence and young adulthood (Harrop & Trower, 2001). Meanwhile, these models typically propose a central role for social adversity in the development of psychotic experiences. Bentall and Fernyhough (2008) have argued that social adversity plays an important role in the development of AH and paranoid thinking. More specifically, they suggest that early traumatic experiences, such as sexual abuse, may be important in the development of AH, while experiences involving discrimination or victimisation, such as bullying, may play a more important role in the development of paranoid thinking. Finally, these models tend to adopt a symptom-specific approach, which allows them to offer different accounts for the development of different psychotic experiences. For example, Freeman (2007) has proposed a model that attempts to explain the development and

maintenance of persecutory delusions, while Beck and Rector (2003) have proposed a model that attempts to explain the development and maintenance of AH. These models sometimes cite common aetiological mechanisms (e.g., both Freeman and Beck & Rector cite negative beliefs about the self as factors that may lead to paranoid thinking and to AH), but a symptom-specific approach allows them to identify different cognitive biases as being important in the development of different psychotic experiences.

Several cognitive-developmental accounts of psychosis propose that (a) poor parental bonding, (b) insecure peer attachment, and (c) reality discrimination biases play a role in the development of psychotic experiences. The results of studies that have investigated these claims are described below. In addition, several unresolved research questions are outlined.

1.2 Parental bonding and psychotic experiences

Parental bonding refers to a person's perceptions of both of their parents' behaviour and attitudes towards them during their childhood, and is assessed through the parental bonding instrument (PBI; Parker, Tupling, & Brown, 1979). Typically, respondents are adults and they are asked to report on their parents' behaviour and attitudes towards them during childhood (i.e., up to the age of 16 years). However, a version of the PBI, suitable for use in adolescents, which assesses current perceptions of parental bonding is also available (Klimidis, Minas, & Ata, 1992). The PBI consists of 12 items which assess levels of care (e.g., "S/he spoke to me in a warm and friendly voice" and "S/he frequently smiled at me") and 13 items which assess the degree of overprotection (e.g., "S/he tried to control everything that I did" and "S/he invaded my privacy"), as factor analytic work has suggested that these two dimensions underpin social relationships (Parker, 1990). Respondents who report having received low levels of care or high levels of overprotection are seen as having received less-optimal caregiving than respondents who report high levels of care or low levels of overprotection.

Six studies have examined reports of parental bonding in patients diagnosed with a psychotic disorder (e.g., schizophrenia, delusional disorder). All of these studies have investigated reports of maternal bonding in this clinical group, with four of the six also examining reports of paternal bonding. Relatively consistent group differences have been reported in terms of maternal and paternal care. Five studies have shown that patients diagnosed with a psychotic disorder report lower levels of maternal care than do control participants (Helgeland & Torgensen, 1997; Melo, Taylor, & Bentall, 2006; Parker, Fairley, Greenwood, Jurd, & Silove, 1982; Rankin, Bentall, Hill, & Kinderman, 2005; Willinger, Heiden, Meszaros, Formann, & Aschauer, 2002). The remaining study (Onstad, Skre, Torgensen, & Kringlen, 1993) reported significant group differences in maternal care in a sub-sample of patients who were compared to an unaffected dizygotic co-twin, but not in a sub-sample of patients who were compared to an unaffected monozygotic co-twin. Three studies have shown that patients diagnosed with a psychotic disorder report lower levels of paternal care than do control participants (Onstad et al., 1993; Parker et al., 1982; Rankin et al., 2005). The remaining study reported group differences in paternal care that approached significance (Melo et al., 2006).

Less consistent group differences have been reported in terms of maternal and paternal overprotection. Two studies have shown that patients diagnosed with a psychotic disorder report higher levels of maternal overprotection than do control participants (Helgeland & Torgensen, 1997; Willinger et al., 2002). Three studies have shown that group differences in maternal overprotection were not statistically significant (Melo et al., 2006; Onstad et al., 1993; Parker et al., 1982). The remaining study reported that while currently paranoid patients reported higher levels of maternal overprotection than did controls, the group difference between remitted paranoid patients and control participants did not reach statistical significance (Rankin et al., 2005). One study has reported that patients diagnosed

with a psychotic disorder report higher levels of paternal overprotection than do control participants (Parker et al., 1982). One study reported group differences in paternal overprotection that were not statistically significant (Melo et al., 2006). One study reported that while currently paranoid patients reported higher levels of paternal overprotection than did controls, the group difference between remitted paranoid patients and control participants did not reach statistical significance (Rankin et al., 2005). Finally, one study reported significant group differences in paternal overprotection in a sub-sample of patients who were compared to a dizygotic co-twin, but not in a sub-sample of patients who were compared to a monozygotic co-twin (Onstad et al., 1993). In sum, patients diagnosed with a psychotic disorder tend to report poorer parental bonding than do control participants, and group differences are most reliably found when comparing reports of maternal and paternal care.

In addition to these studies that have employed clinical samples, two studies have reported associations between poor parental bonding and psychotic experiences in non-clinical samples (Berry, Band, Corcoran, Barrowclough, & Wearden, 2007; Meins, Jones, Fernyhough, Hurndall, & Koronis, 2008), and their findings are broadly consistent with the findings of studies involving clinical populations. Berry et al. (2007) reported that high levels of maternal overprotection and low levels of maternal care were associated with higher levels of unusual experiences, such as magical beliefs (e.g., believing in telepathy) and hallucinatory experiences (e.g., misinterpreting environmental noise as speech), as assessed by the Oxford-Liverpool Inventory of Feelings and Experiences (Mason, Claridge, & Jackson, 1995). Similarly, Meins et al. (2008) reported that low levels of maternal care were associated with higher scores on the suspiciousness/paranoia subscale of the Schizotypal Personality Questionnaire (SPQ; Raine, 1991). However, in contrast to Berry et al., Meins et al. reported that maternal overprotection was not associated with delusion-like beliefs or hallucinatory experiences.

It seems, therefore, that there is an association between reporting poor parental bonding and the presence of psychotic experiences in adulthood. There are a number of reasons why poor parental bonding may lead to the development of psychotic experiences, although this is somewhat under-explained by current models. In terms of paranoid thinking, Bentall, Corcoran, Howard, Blackwood, and Kinderman (2001, p. 1180) have argued that negative early interactions with caregivers may lead to “both a negative attitude toward the self and a tendency to respond to threats to self-esteem by making external attributions” and that these two processes play a direct role in the development of paranoid thoughts. Poor parental bonding is less often cited as a causal factor in the development of AH, although high levels of paternal overprotection are associated with high levels of dissociation (Offen, Thomas, & Waller, 2003), which is thought to play a crucial role in the development of AH (e.g., Longden, Madill, & Waterman, 2012).

Another possibility is that poor parental bonding places a person at risk of being exposed to traumatic experiences—such as sexual abuse (e.g., Hill, Davis, Byatt, Burnside, Rollinson, & Fear, 2000) or bullying (e.g., Rigby, Slee, & Martin, 2007)—and that these may play a direct causal role in the development of psychotic experiences. Strong associations between trauma and psychosis have been reported by a number of studies (see Varese et al., 2012, for a review). For example, in studies that have employed clinical populations, participants with a diagnosis of schizophrenia are more likely to report having experienced some form of trauma than are control participants (e.g., Fisher et al., 2010; Heins et al., 2011; McCabe, Maloney, Stain, Loughland, & Carr, 2012). Similarly, in general population studies, participants who experience AH or delusional beliefs are more likely to report trauma—such as bullying (Lataster et al., 2006), having been placed in institutional care (Bebbington et al., 2004), physical abuse (Shevlin, Dorahy, & Adamson, 2007), and sexual abuse (Bebbington et al., 2011)—than are participants without these experiences. One recent study (Bentall,

Whickham, Shevlin, & Varese, 2012) has suggested that some of these associations may be specific to certain symptoms, with participants who experience AH more likely to report exposure to severe sexual abuse than participants who do not experience AH, and participants who report delusional beliefs more likely to have been placed in institutional care than participants without delusional beliefs.

There is already some evidence that poor parental bonding increases the likelihood that a person will develop psychotic experiences by placing a person at risk of traumatic experiences. This comes from Janssen et al. (2005), who showed, in a general population sample, that the association between low levels of parental care and the presence of psychotic experiences was mediated by childhood trauma. However, Janssen et al.'s measure of childhood trauma was very broad. That is, participants were asked whether they had experienced any kind of (a) emotional, (b) physical, (c) psychological, or (d) sexual abuse before age 16 years, and, if so, were asked to report how often this type of abuse had occurred. It is unclear, therefore, what types of trauma participants may have reported on this measure. For example, as acknowledged by Janssen et al., participants may have been reporting intra-familial abuse, non-familial abuse by an adult, or bullying by peers. Research that examines which of these different forms of adversity mediate the association between poor parental bonding and psychotic experiences would be helpful.

Another way in which poor parental bonding might lead to the development of psychotic experiences is by eliciting high levels of negative affect. A number of studies have reported associations between poor parental bonding and affective problems in adolescence and adulthood (Harris, Brown, & Bifulco, 1986; Rey, 1995). Many models cite affective problems as risk factors for the development of psychotic experiences (e.g., Freeman & Garety, 2003), and this position is supported by a number of longitudinal studies which have reported prospective associations between high levels of negative affect and the onset of

psychotic experiences (Krabbendam et al., 2002). It seems likely, therefore, that this may be one mechanism by which poor parental bonding can lead to the development of psychotic experiences.

1.3 Attachment style and psychotic experiences

Another factor that has been proposed to play a role in the development of psychotic experiences is attachment style. Attachment theory, based upon the work of Bowlby (1969, 1973, 1980) and Ainsworth and colleagues (Ainsworth, 1973; Ainsworth, Blehar, Waters, & Wall, 1978), proposes that infants are biologically predisposed to develop affectional bonds with their caregivers. These bonds, or attachments, are influenced by the way in which caregivers respond to the infant's needs, so that caring, responsive caregiving leads to the development of a secure attachment, while unresponsive caregiving leads to the development of an insecure attachment. In the 1980s, this work was extended to examine individual differences in adults' behaviour in close relationships (e.g., Hazan & Shaver, 1987). Within this literature, attachment style refers to "systematic patterns of expectations, needs, emotions, emotion-regulation strategies, and social behavior that result from... a particular history of attachment experiences, usually beginning in relationships with parents" (Shaver & Mikulincer, 2002, p. 134). This definition suggests that adult attachment style has its roots in early experiences with caregivers. However, there is little evidence that this is the case, with a recent meta-analysis showing that there is no significant stability between attachment security during childhood and attachment style during adulthood (Pinquart, Feußner, & Ahnert, 2013). Despite this problem, Shaver and Mikulincer's definition is useful in that it emphasizes that adult attachment style reflects not just a person's expectations about how they and others behave in close relationships, but also reflects a wider set of strategies for regulating one's emotions.

Unlike the Adult Attachment Interview (AAI; Main, Caplan, & Cassidy, 1985), which assesses a person's current state of mind about the caregiving they received during childhood in terms of the coherence of their discourse about their childhood attachment relationships, attachment style in peer relationships is assessed using self-report questionnaires. These self-report measures of attachment (e.g., the Inventory of Parent and Peer Attachment, Armsden & Greenberg, 1987; the Relationship Questionnaire, Bartholomew & Horowitz, 1991) require participants to report on their thoughts and behaviours in relation to their current attachment relationships (e.g., family relationships, close friendships, and romantic relationships). For example, when completing the Relationship Questionnaire, participants are asked to what extent they agree that a set of statements (e.g., 'It is easy for me to become emotionally close to others. I am comfortable depending on others and having others depend on me. I don't worry about being alone or having others not accept me') describe their behaviour in close relationships with peers and romantic partners. Attachment security assessed using the AAI and attachment style assessed via self-report questionnaires are clearly quite distinct constructs (Roisman, 2009), as reflected in the trivial to small associations between these variables (Roisman et al., 2007). Given that the majority of studies that have examined associations between attachment style and psychotic experiences have employed self-report measures of attachment (Gumley, Taylor, Schwauninger, & MacBeth, 2013), the focus here is on attachment style as assessed through questionnaire-based measures, rather than on attachment security as assessed using the AAI.

Initially, adult attachment style was conceptualised in terms of categories, with individuals being classified as either secure, anxious, or avoidant (e.g., Hazan & Shaver, 1987). However, subsequent work indicated that attachment style was better represented in terms of two dimensions—attachment anxiety and attachment avoidance—along which a person could vary (Bartholomew, 1990; Griffin & Bartholomew, 1994). People who report

high levels of attachment anxiety tend to perceive themselves to be unworthy of love, fear abandonment, and typically attempt to regulate their emotions through a set of hyperactivating strategies (e.g., hypervigilance for threats to attachment relationships, hyperactivation of negative affect; Shaver & Mikulincer, 2002). In contrast, people who report high levels of attachment avoidance tend to have negative beliefs about others, downplay the importance of close relationships, and typically attempt to regulate their emotions through a set of deactivating strategies (e.g., suppression of negative emotions, repression of distressing thoughts and memories; Shaver & Mikulincer, 2002). A number of studies have reported an association between high levels of attachment anxiety and attachment avoidance and the presence of psychotic experiences. For example, in Ponizovsky, Nechamkin, and Rosca's (2007) study, participants with a diagnosis of schizophrenia reported higher levels of attachment anxiety and higher levels of attachment avoidance than did age- and gender-matched controls. Similarly, Mickelson, Kessler, and Shaver (1997) reported that participants with a diagnosis of schizophrenia were more likely to have an avoidant or anxious attachment style than were participants without a diagnosis of schizophrenia. In addition, a number of studies have shown that, within patient groups, higher levels of both attachment anxiety and attachment avoidance are associated with more severe symptoms. For example, Ponizovsky et al. (2007) reported that higher levels of both attachment anxiety and attachment avoidance were associated with more severe psychotic experiences. These findings have been broadly replicated by a number of other studies (e.g., Berry, Barrowclough, & Wearden, 2008; Berry, Wearden, Barrowclough, Oakland, & Bradley, 2012; Ponizovsky, Vitenberg, Baumgarten-Katz, & Grinshpoon, 2013; see Gumley et al., 2013, for a review).

Consistent with these studies involving clinical populations, a number of studies have reported associations between high levels of both attachment anxiety and attachment

avoidance and non-clinical psychotic experiences. For example, several studies have reported associations between high levels of attachment anxiety and paranoid thinking (Berry, Wearden, Barrowclough, & Liversidge, 2006; MacBeth, Schwauninger, & Gumley, 2008; Meins et al., 2008; Pickering, Simpson, & Bentall, 2008; Tiliopolous & Goodall, 2009). All but one of these studies have also reported associations between high levels of attachment avoidance and paranoid thinking (Berry et al., 2006; MacBeth et al., 2008; Pickering et al., 2008; Tiliopolous & Goodall, 2009). In addition, two of these studies have reported associations between hallucination-proneness and high levels of both attachment anxiety and attachment avoidance (Berry et al., 2006; MacBeth et al., 2008).

There are a number of reasons why high levels of attachment anxiety and attachment avoidance may lead to the development of psychotic experiences. In terms of paranoid thinking, high levels of attachment anxiety are associated with negative beliefs about the self (e.g., ‘I am weak and vulnerable’), while high levels of attachment avoidance are associated with negative beliefs about others (e.g., ‘Others are dangerous and are a threat to my safety’; Berry, Barrowclough, & Wearden, 2007). In turn, these beliefs can lead to the development of paranoid thinking (Garety, Kuipers, Fowler, Freeman, & Bebbington, 2001; Bentall & Fernyhough, 2008). High levels of attachment anxiety and attachment avoidance are less often cited as causal factor in the development of AH, and it has been suggested that the associations between AH and both attachment anxiety and attachment avoidance are simply a result of the associations between paranoid thinking and both attachment anxiety and attachment avoidance, with high levels of paranoid thinking increasing the risk that a person will develop hallucinatory experiences (Pickering et al., 2008).

A number of other mechanisms might, however, explain the association between high levels of attachment anxiety and attachment avoidance and psychotic experiences. High levels of attachment anxiety and attachment avoidance are related to a range of interpersonal

problems (e.g., Mallinckrodt, 2000), and it is possible that attachment style may play an indirect role in the development of psychotic experiences through these interpersonal problems. For example, individuals who report high levels of attachment anxiety and attachment avoidance also report high levels of loneliness (e.g., Wei, Russell, & Zakalik, 2005), which is strongly associated with psychosis and the presence of psychotic experiences (e.g., Meltzer et al., 2013). While no model has proposed a causal role for loneliness in the development of psychotic experiences, Hoffman (2007) has hypothesized that social isolation may elicit psychotic experiences, and there is preliminary evidence to support this claim (Murphy, Shevlin, Adamson, & Houston, 2013). Hoffman argues that social isolation gives rise to spurious social experiences, such as perceiving others to be directing their gaze towards you when they are not, and perceiving speech to be present in environmental noise (such as traffic noises or the hum of an air conditioning unit), and that these experiences may form the basis of psychotic symptoms. Given that loneliness and social isolation are closely related to one another (de Jong Gierveld, 1998), loneliness may also elicit psychotic experiences, and could mediate the associations between both attachment anxiety and attachment avoidance and paranoid thinking and the associations between both attachment anxiety and attachment avoidance and AH-proneness.

Another possibility is that the hyperactivating emotion regulation strategies adopted by individuals who experience high levels of attachment anxiety, may play a role in the development of paranoid thinking. Mikulincer, Shaver, and Pereg (2003) have argued that high levels of attachment anxiety are associated with a set of affect regulation strategies that “detect threats in nearly every transaction with the physical and social world and... exaggerate the potential negative consequences of these threats” (p. 85). This is important, as current models of persecutory delusions (e.g., Freeman, 2007) suggest that paranoid thoughts occur when a person experiences an anomalous percept (e.g., a person’s food may taste

strange) and generates a threat-based explanation of that experience (e.g., someone must be trying to poison me), but offer no explanation of why a person might generate a threat-based explanation. Individual differences in attachment anxiety might account for why some people will generate threat-based explanations in response to anomalous percepts and why others will generate more benign explanations. If this is the case, then one would expect attachment anxiety to moderate the association between how often a person experiences anomalous percepts and to what extent they report paranoid thinking.

1.4 Reality discrimination and auditory hallucinations

Reality discrimination refers to the process by which a person distinguishes internal, self-generated events from external, non-self-generated events (Bentall, 1990). Central to several models of AH (e.g., Beck & Rector, 2003; Bentall, 1990; Waters et al., 2012) is the claim that people who experience AH have a tendency to misattribute internal, self-generated events to an external, non-self-generated source. That is, they show biased reality discrimination, so that they make frequent external misattributions, but they rarely make the reverse error (i.e., misattribute an external, non-self-generated event to themselves). Consistent with this argument, a recent meta-analysis has shown that there is a large association between biased reality discrimination and the presence of AH in psychotic patients, and between biased reality discrimination and AH-proneness in non-clinical samples (Brookwell et al., 2013).

Reality discrimination is most commonly assessed using an auditory signal detection task, where participants must try to detect a small amount of speech (typically one second in length) embedded in five seconds of white noise (e.g., Barkus, Stirling, Hopkins, McKie, & Lewis, 2007). In around 20% of trials, this task is relatively easy, as the speech is clearly audible in the white noise. In the remaining trials, judging whether or not the speech is present in the white noise is much more difficult. In around 40% of trials, the speech is

presented at an auditory threshold (determined prior to testing), so that participants will be able to detect the speech in half of these trials. In the remaining 40% of trials, no speech is presented. External misattributions are thought to occur when a participant makes a false alarm (i.e., when participants judge that speech was embedded in the white noise on trials when no speech was presented), as this presumably involves participants misidentifying the internal, self-generated representation of the to-be-detected speech for the external stimulus.

There remain, however, a number of unanswered questions concerning the role reality discrimination biases play in the development of AH. Reality discrimination-based accounts have been criticised for failing to explain why not all cognitive events are misattributed to an external, non-self source (e.g., Gallagher, 2004). This is important as most people who experience AH do not report that they are constant (e.g., McCarthy-Jones, Trauer, MacKinnon, Sims, Thomas, & Copolov, 2012). For example, in Nayani and David's (1996) survey of the phenomenology of AH, only 15% of participants reported that they experienced AH "all of the time" (p. 180). Thus, it seems likely that (a) some factor interacts with reality discrimination biases to result in the onset of an AH, and/or (b) that reality discrimination biases must fluctuate.

The possibility that another factor might interact with reality discrimination to lead to the development of AH makes sense given the nature of the task used to assess reality discrimination. Participants who make external misattributions in the auditory signal detection task do so under situations of high ambiguity (i.e., when it is difficult to determine whether or not the to-be-detected stimulus is present) and they may not make these misattributions under situations of low ambiguity. That is, in the absence of cognitions that are difficult to identify as self-generated, such as intrusive thoughts, participants who have biased reality discrimination may not misattribute any of their cognitions to an external, non-self source (i.e., they may not report hallucinatory experiences). If this is the case, then one

would expect participants who have biased reality discrimination and participants who do not have biased reality discrimination to report similar levels of AH-proneness when both groups report low levels of intrusive thoughts. However, when both of these groups report high levels of intrusive thoughts, one would expect that participants with biased reality discrimination will report greater AH-proneness than participants without biased reality discrimination. Despite a number of suggestions that this might be the case (Bentall, 2003; Beck & Rector, 2003; Bentall et al., 2007), this hypothesis has not been investigated.

The possibility that reality discrimination biases might fluctuate could also account for why AH are not experienced constantly. That is, a person who has a trait-like bias in reality discrimination may not experience AH until that bias is exacerbated by some other factor. Variables that have been identified as ‘triggers’ of AH would seem to be good candidates for factors that might cause fluctuations in reality discrimination. Patients who experience AH have cited a wide range of negative emotions—such as sadness, anger, fear, and anxiety—as triggers of AH (Nayani & David, 1996; Tarrier, 1987). The importance of anxiety in the day-to-day onset of AH has been supported by an experience sampling study, which reported that feelings of anxiety often preceded the onset of an AH (Delespaul, de Vries, & van Os, 2002). Being alone and feelings of loneliness have also been identified by patients as triggers of AH (Nayani & David, 1996; Tarrier, 1987) and, as discussed above, there are theoretical reasons to believe that feelings of loneliness might trigger AH. Research that examines whether negative emotions, including loneliness, modulate reality discrimination biases would be of interest.

1.5 Socio-economic factors and psychotic experiences

Alongside parental bonding, attachment style, and reality discrimination, broad socio-economic factors appear to play an important role in the development of psychotic experiences (van Os, Kenis, & Rutten, 2010). For example, a number of studies have reported

that rates of psychosis are elevated in areas characterised by high levels of social deprivation (e.g., high levels of unemployment, overcrowding, and low incomes) than in more affluent areas (e.g., Croudace, Kayne, Jones, & Harrison, 2000; Koppel & McGuffin, 1999).

Consistent with these findings, it has been reported that psychosis patients are more likely to have been born to fathers of a low socio-economic group (i.e., a social class of IV or V, based on his occupation) than are control participants (Harrison, Gunnell, Glazerbrook, Page, & Kwiecinski, 2001), and that unemployment prospectively predicts the diagnosis of a psychotic disorder 15 years later (Agerbo, Byrne, Eaton, & Mortensen, 2004). These factors are typically not the focus of psychological models of psychotic experiences, and are not the focus of this thesis. However, where possible, some measure of socio-economic status should be included as a covariate in studies investigating the psychological predictors of psychotic experiences, given that some psychological variables (e.g., low self-esteem) are related to socio-economic factors (Richman, Clark, & Brown, 1985).

1.6 Chapter summary and outline of thesis

In this General Introduction, a number of unanswered research questions about the roles that poor parental bonding, insecure peer attachment, and biased reality discrimination play in the development of psychotic experiences have been described. Across five studies, several of these research questions will be addressed in this thesis, as explained below.

Recent research has suggested that examining the development of non-clinical psychotic experiences (referred to as psychotic-like experiences, or PLEs; Kelleher & Cannon, 2011) can inform our understanding of how the AH and paranoid thinking reported by psychotic patients develop (e.g., Kaymaz et al., 2012; Kelleher & Cannon, 2011). The studies presented in this thesis have examined these research questions by investigating the predictors of PLEs in non-clinical adolescent and adult samples.

The first three studies of this thesis address questions related to the roles played by parental bonding and attachment style in the development of PLEs. In Study 1, a question concerning the role parental bonding plays in the development of PLEs was addressed. At present, it is unclear why poor parental bonding increases a person's risk of developing psychotic experiences. However, poor parental bonding is associated with exposure to traumatic experiences, such as bullying (Rigby et al., 2007), and with experiencing symptoms of depression (Rey, 1995). Meanwhile, both exposure to bullying (van Dam et al., 2012) and high levels of negative affect (Krabbendam et al., 2002) appear to play a role in the development of psychotic experiences. It is plausible, therefore, that exposure to bullying and negative affect mediate the associations between poor parental bonding and PLEs. Study 1 examined whether this was the case.

In Study 2, a question concerning the roles attachment anxiety and attachment avoidance play in the development of PLEs was addressed. As discussed above, high levels of attachment anxiety and attachment avoidance may indirectly lead to the development of psychotic experiences by placing a person at risk of interpersonal problems, such as loneliness (Wei et al., 2005), and these interpersonal problems may play a direct role in the development of psychotic experiences. Thus, in Study 2, the extent to which loneliness mediates the association between attachment style and PLEs was examined.

In Study 3, a question concerning the role high levels of attachment anxiety play in the development of paranoid thinking was investigated. It has been argued that people who report high levels of attachment anxiety perceive innocuous events to be indicators of social threat (Mikulincer et al., 2003). This sort of bias may be important in the development of persecutory delusions, as paranoid thoughts are claimed to be triggered when a person experiences an anomalous percept and generates a threat-based explanation for that percept (Freeman, 2007). Individual differences in attachment anxiety may thus moderate the

association between anomalous percepts and paranoid thinking, and this possibility was examined in Study 3.

The aim of the fourth and fifth studies reported here was to examine two questions related to reality discrimination. First, it has been proposed that a person who has biased reality discrimination abilities *and* who experiences intrusive thoughts should be especially prone to AH (Bentall, 2003; Bentall et al., 2007). Study 4 investigated if this was the case by examining whether reality discrimination abilities moderate the association between intrusive thoughts and AH-proneness in a non-clinical sample of young adults. Second, given that many voice-hearers do not experience constant AH (e.g., McCarthy-Jones et al., 2012), it seems likely that a person's reality discrimination abilities must fluctuate. One possibility is that emotion may modulate a person's reality discrimination abilities, as voice-hearers often report emotional changes prior to the onset of voices (e.g., Nayani & David, 1996). In Study 5, the possibility that mood can modulate reality discrimination was examined in a non-clinical sample of young adults.

Together, the findings of these studies addressed a number of questions relevant to current cognitive-developmental accounts of psychotic experiences. Primarily, they investigated to what extent a variety of social (e.g., poor parental bonding, exposure to bullying), emotional (e.g., negative affect), and cognitive factors (e.g., biased reality discrimination) mediate and/or moderate one another to place a person at risk of developing PLEs. Establishing these mediation and/or moderation effects should allow for the formation of more precise models of the developmental trajectories of psychotic experiences.

Chapter 2

Study 1: Associations between parental bonding, negative affect, bullying, and psychotic-like experiences in a non-clinical sample of adolescents

In Study 1, associations between parental bonding and PLEs were examined in a non-clinical sample of adolescents. Reliable associations have been reported between some aspects of parental bonding (e.g., maternal care) and psychotic experiences, but it remains unclear why this relation exists. One possibility is that poor parental bonding increases the likelihood that a person will experience traumatic interpersonal experiences (e.g., bullying) and affective problems, and that these two factors will play a direct role in the development of psychotic experiences. Thus, Study 1 examined whether exposure to bullying and negative affect mediated the associations between parental bonding and PLEs.

2.1 Abstract

This study examined whether bullying and negative affect mediated the associations between parental bonding and psychotic-like experiences (PLEs) in a sample of adolescents. Participants were 318 schoolchildren (mean age = 14.42 years; 58% girls) who completed a questionnaire pack containing measures of current parental bonding, bullying, affect, paranoid thinking, and hallucination-proneness. Low levels of care, high levels of overprotection, high levels of negative affect, and high levels of bullying were positively associated with PLEs. Regression analyses showed that maternal care, but not overprotection, was an independent predictor of both paranoid thinking and hallucination-proneness. Bootstrapping analyses revealed that the associations between maternal care and paranoid thinking, and between maternal care and hallucination-proneness, were mediated by bullying and negative affect. These results suggest that low levels of maternal care may lead to PLEs by eliciting negative affect and by placing a child at risk of subsequent social adversity.

2.2 Introduction

Over the past 10 years, researchers (e.g., van Os, Hanssen, Bijl, & Vollebergh, 2001) have demonstrated that a relatively large proportion of the population report psychotic-like experiences (PLEs), that is, psychotic symptoms (e.g., hallucinatory experiences and paranoid thinking) that occur in the absence of a psychotic illness (Kelleher & Cannon, 2011). PLEs appear to be especially common in adolescence, with around 20% of younger adolescents reporting a PLE in the past year, with that figure falling to around 7% for older adolescents (Kelleher et al., 2012). Adolescents who report PLEs are at an increased risk of subsequently being diagnosed with a psychotic disorder (Poulton et al., 2000; although the majority will not, see Dominguez, Wichers, Lieb, Wittchen, & van Os, 2011). The study of PLEs therefore allows researchers to examine the factors that influence the development of psychosis without the confounding effects of medication and illness chronicity that affect studies with clinical populations. In addition to being a risk factor for psychosis, the presence of PLEs is associated with poorer mental health more generally. For example, adolescents who report high levels of PLEs are more likely to report self-harm and suicidal ideation (Nishida et al., 2008; Polanczyk et al., 2010) and are much more likely to have a diagnosable mental health problem (Kelleher et al., 2012; Scott et al., 2009) in comparison to their peers. Careful research into the development of PLEs thus appears to be important not just in understanding the development of psychosis, but also in understanding the mental health of adolescents more generally.

A number of studies have demonstrated that individuals diagnosed with a psychotic disorder report lower levels of parental care and higher levels of parental overprotection (i.e., poorer parental bonding) than do controls (Helgeland & Torgensen, 1997; Melo et al., 2006; Onstad et al., 1993; Parker et al., 1982; Rankin et al., 2005; Willinger et al., 2002). More recently, studies have shown that there is also an association between sub-optimal parental

bonding and PLEs. For example, Berry et al. (2007) reported that high levels of maternal overprotection and low levels of maternal care were associated with higher levels of unusual experiences, such as magical beliefs and hallucinatory experiences. Similarly, Meins et al. (2008) found that low levels of maternal care predicted higher scores on the suspiciousness/paranoia subscale of the schizotypal personality questionnaire (Raine, 1991). However, in contrast to Berry et al. (2007), Meins et al. reported that maternal overprotection was not associated with any type of PLE. Further work is thus required to clarify the associations between parental bonding and PLEs. This is especially true in adolescents, as no studies have examined these associations in this population (although Polanczyk et al., 2010, have reported a positive association between maternal expressed emotion and PLEs in 12-year-olds).

Another outcome associated with negative parenting experiences is bullying. Children and adolescents who are bullied—that is, who are the victims of intentional acts of aggression that are repetitive and that involve an imbalance of power (Olweus, 1993)—tend to report poorer relationships with their parents than do their peers (Bowers, Smith, & Binney, 1994; Rigby, 1993). More precisely, it has been shown that mothers of bullied children tend to be more overprotective than those of non-bullied children, and this has been demonstrated for both child-rated (Finnegan, Hodges, & Perry, 1998) and maternal-rated levels of overprotection (Georgiou, 2008). Associations between bullying and more problematic parenting have also been reported. For example, it has been shown that maltreated children are more likely to be bullied than non-maltreated children (Shields & Cicchetti, 2001), and that adolescents who are in contact with child protection services are more likely to be bullied than their peers (Mohapatra et al., 2010).

As well as being associated with negative parenting experiences, bullying is robustly related to the presence of PLEs. A number of cross-sectional studies have reported that both

hallucination-proneness and paranoid thinking are associated with being bullied (Campbell & Morrison, 2007; Lataster et al., 2006; Nishida et al., 2008; Oshima et al., 2010). Importantly, these studies have been followed by longitudinal work, which has shown that bullying predicts the development of PLEs in adolescents (Arsenault et al., 2011; Mackie, Castellanos-Ryan, Conrod, 2011; Schreier et al., 2009), even after controlling for baseline psychopathology, IQ, socio-economic status, and genetic risk of psychosis. It has been argued that these results suggest a possible causal relation between bullying and PLEs (Schreier et al., 2009). Given these associations, it seems possible that bullying may mediate the associations between parental bonding and PLEs.

Another plausible mediator of the associations between parental bonding and PLEs is negative affect. Numerous studies have shown that low parental care is associated with the development of high levels of negative affect (e.g., Harris et al., 1996; Rey, 1995) and that negative affect is important in the development of both clinical psychosis (Jones et al., 1994) and PLEs (Kelleher & Cannon, 2011). There is already evidence that negative affect mediates the effects of negative parenting experiences on these outcomes in adults. For example, it has been shown that the association between psychosis in adulthood and being taken into local authority care as a child was reduced (from an odds ratio of 10.71 to one of 2.5) once current levels of depression had been controlled (Bebbington et al., 2004). Thus, the present study set out to examine, in a sample of adolescents, whether associations between parental bonding and PLEs were mediated by negative affect and experiences of bullying.

2.3 Methods

2.3.1 Participants

Participants were 318 (132 boys, 181 girls, five participants did not report sex) 13- to 15-year-olds ($M = 14.42$ years; $SD = 6.18$), recruited from three schools in north-east

England. Letters explaining the study, and asking consent for the child's participation, were sent to the parents/carers of 646 pupils, giving a participation rate of 49.23%.

2.3.2 *Materials and procedure*

Testing took place during normal school lessons (a personal, social, health, and economic education lesson, or an extended registration period). Pupils were asked to complete the questionnaire pack without allowing others to see their responses, but were allowed to ask the researcher to explain any questions that they did not understand. Participants completed the questionnaires in the order described below.

Parental bonding. Parental bonding was assessed using the brief, current version of the parental bonding instrument (PBI-BC; Klimidis et al., 1992). This scale, designed for use with adolescents, consists of eight items that measure current perceptions of parental behaviour. Four items assess perceived care, and four assess perceived overprotection, with participants reporting on a 4-point Likert scale how often their carer behaves in a certain way (1 = *rarely or never*, 4 = *almost always*). Participants completed the PBI-BC twice – once for a maternal carer, and once for a paternal carer. Participants were allowed to report who their main maternal and paternal carers were (i.e., it was not assumed that the participant's biological mother/father was their main maternal/paternal carer). Scores for each subscale range from 4 to 16, with higher scores reflecting greater levels of care or of overprotection.

For the purposes of this study, one item of the PBI-BC was simplified. Item 4 originally read, "He/she seems NOT affectionate (emotionally cold) to me". This was changed to, "He/she seems cold to me". The maternal ($\alpha = .78$) and paternal ($\alpha = .79$) care subscales had acceptable levels of internal reliability. The maternal ($\alpha = .64$) and paternal overprotection ($\alpha = .57$) subscales had levels of internal reliability lower than is normally acceptable (i.e., $\alpha > .7$). However, for brief scales, α -values around 0.6 can be considered acceptable (Loewenthal, 1996).

Bullying. Severity of bullying was assessed using four questions derived from the definition of bullying used by Bowes, Maughan, Caspi, Moffitt, and Arseneault (2010). Participants were asked how frequently in the past year another child at school (a) said mean or hurtful things, (b) ignored you or made sure you were left out of things, (c) hit, kicked, pushed you, or locked you in a room, and (d) told lies or spread rumours about you. Response options were: never; once or twice; two or three times per month; about once a week; several times per week. Response options were scored on a range of 1 to 5, so that total scores ranged from 4 to 20, with higher scores reflecting more severe bullying. The scale had acceptable levels of internal reliability ($\alpha = .77$).

Hallucination-proneness. Hallucination-proneness was assessed using a recently revised version of the Launay-Slade hallucination scale (RHS; Launay & Slade, 1981; McCarthy-Jones & Fernyhough, 2011). This 5-item version contains only items referring to auditory experiences (e.g., “I hear a voice speaking my thoughts aloud”), and has better psychometric properties than previous versions of the Launay-Slade scale (see McCarthy-Jones & Fernyhough, 2011). Participants indicate how much each item applies to them on a 5-point Likert scale (0 = *certainly does not apply*, 4 = *certainly applies*), so that scores can range from 0 to 20, with higher scores reflecting greater hallucination-proneness. In this sample, the scale had acceptable levels of internal reliability ($\alpha = .82$).

Paranoid thinking. Paranoid thinking was assessed using the persecution items from the persecution and deservedness scale (PADS; Melo, Corcoran, Shyrane, & Bentall, 2009). This scale consists of ten statements, which describe aspects of paranoid thinking (e.g., “There are times when I worry that others might be plotting against me”). Participants are asked to indicate to what extent they believe each item on a 5-point Likert scale (0 = *certainly false*; 4 = *certainly true*), so that scores can range from 0 to 40, with higher scores reflecting higher levels of paranoid thinking. Given the age of the present sample, two of the PADS

items were revised so that they were easier to comprehend. Item 3 was revised from, “My friends/others often tell me to relax and stop worrying about being deceived or harmed” to, “My friends/others often tell me to relax and stop worrying about being lied to or harmed”. Item 6 was revised from, “Sometimes, I just know that people are talking critically about me” to, “Sometimes, I just know that people are criticizing me”. In this sample, the scale had acceptable levels of internal reliability ($\alpha = .89$).

Positive and negative affect. Positive and negative affect was assessed using the revised version of the Positive and Negative Affect Schedule, which is suitable for adolescents (Joiner, Catanzaro, & Laurent, 1996). This scale consists of ten words that describe positive affect (PA; e.g., excited) and ten that describe negative affect (NA; e.g., scared). Participants are asked to indicate to what extent they generally (i.e., on average, rather than over a specific time-frame) feel each emotion on a 5-point Likert scale (1 = *very slightly or not at all*, 5 = *extremely*), so that scores on each subscale range from 10 to 50, with higher scores reflecting higher levels of positive or of negative affect. The NA subscale had acceptable levels of internal reliability in this sample ($\alpha = .85$). The PA subscale, however, did not ($\alpha = .66$).

2.4 Results

2.4.1 Descriptive statistics and preliminary analyses

Descriptive statistics are presented in Table 2.1. To examine the associations between gender and the predictor and outcome variables, a series of *t*-tests were performed, investigating differences between boys and girls. Not all participants provided complete data. Hence, the degrees of freedom associated with the *t*-tests reported here are not uniform. Sex differences in parental bonding, bullying, and hallucination-proneness were not significant (all $p > .10$). However, girls reported lower levels of positive affect ($M = 30.91$, $SD = 6.25$) than did boys ($M = 32.77$, $SD = 7.01$), $t(303) = 2.44$, $p = .015$, $d = 0.28$. Girls reported higher

Table 2.1 Descriptive statistics for, and correlations between, measures of parental bonding, bullying, affect, hallucination-proneness, and paranoid thinking

Variable	Mean (SD)	Min.–Max.	2	3	4	5	6	7	8	9
1. Maternal care	13.68 (2.36)	5–16	-.45*	.49*	-.24*	-.20*	.26*	-.38*	-.24*	-.29*
2. Maternal overprotection	7.07 (2.16)	4–16		-.20*	.54*	.26*	-.17	.32*	.18*	.21*
3. Paternal care	12.95 (2.75)	6–16			-.41*	-.11	.27*	-.20*	-.12	-.17
4. Paternal overprotection	6.55 (1.99)	4–14				.14	-.25*	.16	.07	.16
5. Bullying	7.86 (2.96)	4–20					-.07	.46*	.34*	.57*
6. Positive affect	31.69 (6.65)	15–49						-.08	-.10	-.08
7. Negative affect	20.48 (7.06)	10–46							.49*	.65*
8. Hallucination-proneness	6.59 (5.20)	0–20								.58*
9. Paranoid thinking	14.35 (9.30)	0–39								

* $p < .0014$

levels of negative affect ($M = 22.49$, $SD = 7.57$) than did boys ($M = 19.61$, $SD = 6.60$), $t(305) = 3.47$, $p = .001$, $d = 0.39$. Girls also reported higher levels of paranoid thinking ($M = 16.57$, $SD = 9.56$) than did boys ($M = 13.39$, $SD = 8.94$), $t(308) = 2.97$, $p = .003$, $d = 0.34$.

Correlations between variables are also presented in Table 2.1. A Bonferroni correction was applied to adjust for the number of correlations performed, meaning that a significance level of $\alpha' = .0014$ was employed. Not all participants provided complete data. As a result, the size of N varies from 298–317 for the correlational analyses. Lower levels of parental care, higher levels of overprotection, more severe bullying, and higher levels of negative affect were all associated with higher levels of PLEs. Given the non-significant associations between PLEs and paternal care, paternal overprotection, and positive affect, these three variables were not included as predictors in subsequent regression analyses.

2.4.2 Predictors of hallucination-proneness

Linear regression was conducted to identify independent predictors of hallucination-proneness. As shown in Table 2.2, age, sex, maternal care, and maternal overprotection were entered as predictors in the first block, with bullying and negative affect entered in the second block. Collinearity diagnostics for this regression were satisfactory (minimum tolerance = .63; average VIF = 1.30), and residuals appeared to be both independent and normally distributed. The initial model was significant, $F(4, 298) = 5.80$, $p < .001$, adjusted $R^2 = .06$. In this model, only maternal care was a significant predictor of hallucination-proneness. Inclusion of bullying and negative affect improved the model, $F(2, 296) = 38.12$, $p < .001$, $\Delta R^2 = .19$. In this model, bullying and negative affect were significant predictors of hallucination-proneness.

2.4.3 Predictors of paranoid thinking

A second linear regression analysis was conducted to identify independent predictors of PADS score. As shown in Table 2.3, age, sex, maternal care, and maternal overprotection

Table 2.2 Summary of hierarchical regression analysis for hallucination-proneness

Variable	B	SE B	β
<i>Block 1</i>			
Age	0.05	0.05	.06
Sex	0.77	0.62	.07
Maternal care	-0.43	0.14	-.19**
Maternal overprotection	0.24	0.22	-.11
<i>Block 2</i>			
Age	0.06	0.04	.07
Sex	-0.30	0.58	-.03
Maternal care	-0.09	0.14	-.04
Maternal overprotection	-0.02	0.14	-.01
Bullying	0.25	0.10	.14*
Negative affect	0.31	0.05	.41***

* $p < .05$, ** $p < .01$, *** $p < .001$

were entered as predictors in the first block, with bullying and negative affect entered in the second block. Collinearity diagnostics for this regression were satisfactory (minimum tolerance = .64; average VIF = 1.30), and residuals appeared to be both independent and normally distributed. The initial model was significant, $F(4, 299) = 10.55$, $p < .001$, adjusted $R^2 = .11$. In this model, only sex and maternal care were significant predictors of paranoid thinking. Inclusion of bullying and negative affect improved the model, $F(2, 297) = 123.39$, $p < .001$, $\Delta R^2 = .40$. In this model, sex, bullying, and negative affect were significant predictors of paranoid thinking.

Table 2.3 Summary of hierarchical regression analysis for paranoid thinking

Variable	B	SE B	β
<i>Block 1</i>			
Age	-0.00	0.08	-.00
Sex	3.59	1.05	.19**
Maternal care	-1.02	0.24	-.26***
Maternal overprotection	0.39	0.26	-.09
<i>Block 2</i>			
Age	0.02	0.06	.02
Sex	1.69	0.81	.09*
Maternal care	-0.28	0.19	-.07
Maternal overprotection	-0.27	0.20	-.07
Bullying	1.08	0.14	.35***
Negative affect	0.60	0.07	.46***

* $p < .05$, ** $p < .01$, *** $p < .001$

It could be argued that three of the PADS items (“There are times when I worry others might be plotting against me”, “Sometimes I just know that people are criticizing me”, and “I believe that some people want hurt me deliberately”) assess perceptions of bullying, rather than paranoid thinking. To address this issue, the above regression analysis was conducted again, with scores on a 7-item version of the PADS (i.e., with those three items excluded) used as the outcome variable. The pattern of results for this analysis was identical to that for the original analysis.

2.4.4 Mediation analyses

Given that maternal care was the only parental bonding variable that was a significant predictor of either hallucination-proneness or paranoid thinking, mediation analyses were only carried out for this predictor. Mediation was assessed by examination of the indirect effects of maternal care on each PLE via negative affect and bullying. This was done using a bootstrapping sampling procedure, with 5,000 samples, conducted by INDIRECT, an SPSS macro developed by Preacher and Hayes (2008). Variables were identified as significant mediators if the 95% confidence intervals of their associated point estimates for indirect effects did not cross zero (Preacher & Hayes, 2004). The direct effect of maternal care on hallucination-proneness was not significant, $B = -0.04$, $SE = 0.12$, $t = -0.39$, $p = .70$. As shown in Table 2.4, there were significant indirect effects of maternal care on hallucination-proneness through negative affect and bullying. Similarly, the direct effect of maternal care on paranoid thinking was not significant, $B = -0.14$, $SE = 0.17$, $t = -0.85$, $p = .39$. As shown in Table 2.4, there were significant indirect effects of maternal care on paranoid thinking through negative affect and bullying. As with the regression analysis reported above, this bootstrapping sampling procedure was repeated with scores on a 7-item version of the PADS used as the outcome variable. Again, the pattern of results for this analysis was identical to that for the original analysis. These results suggest that the associations between maternal care and both forms of PLE are fully mediated by negative affect and bullying.

2.4.5 Controlling for co-occurring PLEs

It has been argued that when examining the predictors of hallucination-proneness, one should control for the co-occurrence of paranoid thinking, and vice versa (Pickering et al., 2008). This is because these two PLEs frequently co-occur and so an association between a predictor and one PLE, say hallucination-proneness, may simply be a result of the association

Table 2.4 Mediation of the association between maternal care and PLEs by bullying and negative affect

	Hallucination-proneness			Paranoid thinking		
	Total indirect effect			Total indirect effect		
	Point	BC 95% CI		Point	BC 95% CI	
	estimate	Lower	Upper	estimate	Lower	Upper
Bullying	-0.07	-0.15	-0.02	-0.29	-0.49	-0.13
Negative affect	-0.38	-0.57	-0.24	-0.73	-1.03	-0.48

BC 95% CI = Bias corrected 95% confidence interval

between that predictor and another PLE, say paranoid thinking. Thus, the analyses above were repeated when controlling for co-occurring PLEs.

2.4.5.1 Predictors of hallucination-proneness when controlling for co-occurring paranoia

Linear regression was conducted to identify independent predictors of hallucination-proneness. Age, sex, and paranoid thinking were entered as predictors in the first block, maternal care, and maternal overprotection were entered as predictors in the second block, with bullying and negative affect entered in the third block. Collinearity diagnostics for this regression were satisfactory (minimum tolerance = .49; average VIF = 1.52), and residuals appeared to be both independent and normally distributed. The initial model was significant, $F(3, 299) = 43.40, p < .001$, adjusted $R^2 = .30$. In this model, paranoid thinking was a significant predictor of AH-proneness. Inclusion of maternal care and maternal overprotection did not improve the model, $F(5, 297) = 26.72, p < .001, \Delta R^2 = .01$. Inclusion of bullying and negative affect improved the model, $F(7, 295) = 21.44, p < .001, \Delta R^2 = .03$. In this model, paranoid thinking and negative affect were significant predictors of hallucination-proneness.

2.4.5.2 Predictors of paranoid thinking when controlling for co-occurring AH-proneness

Linear regression was conducted to identify independent predictors of hallucination-proneness. Age, sex, and AH-proneness were entered as predictors in the first block, maternal care, and maternal overprotection were entered as predictors in the second block, with bullying and negative affect entered in the third block. Collinearity diagnostics for this regression were satisfactory (minimum tolerance = .55; average VIF = 1.35), and residuals appeared to be both independent and normally distributed. The initial model was significant, $F(3, 299) = 46.21, p < .001$, adjusted $R^2 = .31$. In this model, gender and AH-proneness were significant predictors of paranoid thinking. Inclusion of maternal care and maternal overprotection did not improve the model, $F(5, 297) = 32.23, p < .001, \Delta R^2 = .03$. In this model, gender, AH-proneness, and maternal care were significant predictors of paranoid thinking. Inclusion of bullying and negative affect improved the model, $F(7, 295) = 55.71, p < .001, \Delta R^2 = .22$. In this model, gender, AH-proneness, bullying, and negative affect were significant predictors of paranoid thinking.

2.4.5.3 Mediation analysis

Given that maternal care was not an independent predictor of hallucination-proneness when paranoid thinking had been controlled for, mediation analysis was not carried out for this outcome variable. As in the original analysis, mediation was assessed by examination of the indirect effects of maternal care on paranoid thinking via negative affect and bullying, using a bootstrapping sampling procedure, conducted by INDIRECT (Preacher & Hayes, 2008). The direct effect of maternal care on paranoid thinking was not significant, $B = -0.14, SE = 0.17, t = -0.85, p = .39$. As in the original analysis, there were significant indirect effects of maternal care on paranoid thinking through negative affect and bullying.

2.5 Discussion

The present results suggest that low levels of maternal care are associated with both hallucination-proneness and paranoid thinking in adolescents, and that these associations are mediated by both negative affect and bullying. These findings are consistent with research that has reported associations between poor parental bonding and psychosis (e.g., Helgeland & Torgersen, 1997; Onstad et al., 1993), between maternal expressed emotion and PLEs in adolescents (Polanczyk et al., 2010), and between poor parental bonding and PLEs in non-clinical samples of adults (Berry et al., 2007; Meins et al., 2008). The current findings also suggest that maternal care is a better predictor of the development of PLEs than maternal overprotection, which is consistent with the data reported by Meins et al. (2008). Taken together, these studies provide support for approaches that propose a role for parental care and/or attachment experiences in the development of psychotic experiences (e.g., Bentall et al., 2007) and for approaches that suggest that PLEs are associated with the same risk factors as psychosis (e.g., Kelleher & Cannon, 2011). The present results, however, also identify two mechanisms—the creation of negative affect and exposure to bullying—that help to explain why low levels of care lead to the development of PLEs, and perhaps to the development of clinically-relevant psychotic experiences.

It should be noted, however, that when controlling for paranoid thinking, there was no association between maternal care and hallucination-proneness. This data analysis strategy is recommended by, for example, Pickering et al. (2008), so that one can demonstrate specific links between predictor variables and certain types of PLEs. However, this strategy has recently been criticised by van Nierop et al. (2014), who claim that much more complex statistical analysis is required to demonstrate specific associations between predictor variables and PLEs. Thus, the value of this approach remains a matter of debate.

The mediating role played by negative affect is consistent with a number of lines of research. First, this finding is consistent with previous reports of relations between low levels of parental care and affective problems in adolescence (Rey, 1995) and adulthood (Harris et al., 1986), and with approaches that emphasize the importance of affective problems in the development of psychotic experiences (Freeman & Garety, 2003). More specifically, the present data are consistent with reports that negative affect partially mediates the association between indicators of poor parent–child relationships (e.g., being taken into local authority care) and the development of psychotic experiences (Bebbington et al., 2004). The present data, however, extend these findings into an adolescent sample.

Similarly, the mediating role of bullying is consistent with research that has suggested that poor parent–child relationships place children at risk of being bullied (e.g., Shields & Cicchetti, 2001), and with a number of studies that have reported associations between being bullied and the development of PLEs (Arsenault et al., 2011; Mackie et al., 2011; Schreier et al., 2009). This body of work linking bullying to the development of PLEs supports approaches that emphasize the role experiences of victimization—such as bullying, but also experiences like physical abuse and discrimination—play in the development of psychosis (e.g., Bentall & Fernyhough, 2008). More generally, the associations between bullying and PLEs reported here are consistent with a growing body of evidence demonstrating the negative effects of bullying on mental health (e.g., Arsenault, Bowes, & Shakoor, 2010; Reijntjes, Kamphuis, Prinzie, & Telch, 2010).

In addition, the mediating role of bullying is consistent with a number of studies that have shown that the associations between difficult parent–child relationships and mental health problems in adolescence and adulthood are mediated by subsequent traumatic experiences. In terms of the development of psychosis, this has been shown by Janssen et al. (2005), who reported that the relation between low levels of parental care and the onset of

psychotic experiences was mediated by childhood trauma. Similar findings have been reported for other types of psychopathology, where the effects of parental abuse are mediated by revictimization in adolescence and adulthood (e.g., Lindhorst, Beadnell, Jackson, Fieland, & Lee, 2009; Stein, Leslie, & Nyamathi, 2002). It seems possible, therefore, that some of the experiences of poor parenting that have been identified as predictors of psychosis and other forms of psychopathology confer an increased risk of these outcomes by elevating the chances that an individual will experience social adversity and/or trauma, which might play a more direct role in the development of mental health problems. Future research should assess whether this is the case.

These findings have several practical implications. These data provide further support for the importance of interventions that aim to prevent or reduce levels of school-based bullying (e.g., Andreou, Didaskalou, & Vlachou, 2007; Fonagy et al., 2009; King, Vidourek, Davis, & McClellan, 2002). Several reviews have suggested that anti-bullying interventions are effective in reducing the prevalence of bullying (e.g., Ttofi & Farrington, 2011), although these effects are small and often are not maintained at follow-up (Barbero, Hernandez, Esteban, & Garcia, 2012), and there is debate as to whether these effects are of practical value (Ferguson, San Miguel, Kilburn, & Sanchez, 2007; Smith, Schneider, Smith, & Ananiadou, 2004). Importantly, given the present data, some interventions have been shown not only to reduce the frequency of incidents of bullying, but also appear to reduce levels of negative affect. For example, King et al. (2002) reported that students who were admitted to a mentoring programme were less likely to be bullied than control children, and were less likely to experience symptoms of depression. Other studies have also shown that pupils report reductions in bullying (e.g., Elledge, Cavell, Ogle, & Newgent, 2010) and in negative affect (e.g., Jackson, 2002) following a mentoring intervention. If future research replicates these effects, then the present results indicate that these interventions may be particularly effective

in preventing the development of PLEs, in comparison to curriculum-based interventions that involve raising pupils' awareness of bullying and its negative effects (e.g., Andreou et al., 2007). In addition, the present findings suggest that children who receive low levels of maternal care, in particular, might benefit from interventions such as mentoring. Previous reports have typically focused on factors such as age and gender (e.g., Karcher, 2008) in predicting which pupils will gain most from mentoring interventions. The present results indicate that future intervention studies might specifically target children and adolescents who perceive their relationships with their care-givers to be problematic, as they appear to be an at-risk group.

This study suffered from a number of limitations. First, it is possible that some of the PADS items could be seen as items that assess perceived bullying, rather than paranoid thinking. This is a problem as it might have inflated associations between bullying and paranoid thinking, and so made it more likely that bullying would mediate the association between maternal care and paranoid thinking. This issue does not, however, seem to have been a major problem here, as identical patterns of results were found when we repeated the relevant regression and mediation analyses using a version of the PADS that did not include the potentially problematic items.

Other limitations are that the study relied on information from a single source and was cross-sectional, meaning that some of the associations reported here may be inflated due to shared method variance, and that no conclusions can be drawn about the causal relations we have proposed here. However, previous studies that have employed multiple informants and/or longitudinal designs support our conclusions (e.g., Georgiou, 2008; Jones et al., 1994; Schreier et al., 2009). That said, further research that examines the associations reported here and employs a longitudinal design, involving multiple informants (e.g., parents, teachers, and peers), is required.

Chapter 3

Study 2: Associations between attachment style, loneliness, and psychotic-like experiences in a non-clinical sample of adults

The two studies reported in Chapters 3 and 4 move away from examining associations between parental bonding and PLEs, and address questions concerning the ways in which attachment anxiety and attachment avoidance relate to psychotic experiences. In Study 2, associations between attachment anxiety, attachment avoidance, loneliness, and PLEs were examined in a non-clinical sample of adults. While it has been suggested that social isolation—a variable closely related to loneliness—may play a direct role in the development of psychotic experiences, most models suggest that attachment anxiety and attachment avoidance play indirect roles in the development of psychotic experiences, through a number of other variables. Thus, Study 2 aimed to establish whether the associations between PLEs and attachment anxiety and attachment avoidance were mediated by loneliness.

3.1 Abstract

Attachment style and loneliness appear to be closely related traits, and both may play a role in the development of psychotic-like experiences (PLEs). The present study examined associations between attachment style and PLEs in a non-clinical sample of adults, and investigated whether loneliness mediated these associations. Participants were 368 adults (18-45 years) who completed an online questionnaire battery. Hallucination-proneness was not associated with attachment anxiety, attachment avoidance, or loneliness. After controlling for demographic variables, recent cannabis use, negative affect, and hallucination-proneness, attachment anxiety and attachment avoidance were both significant predictors of paranoid thinking. Loneliness partially mediated the associations between both attachment variables and paranoid thinking. These findings suggest that attachment style is specifically related to paranoid thinking, and that the development of loneliness may be one way in which high levels of attachment anxiety and attachment avoidance lead to paranoid thinking.

3.2 Introduction

Traditionally, adult attachment style was conceptualised in terms of categories, with individuals falling into one of three groups—secure, anxious, or avoidant (e.g., Hazan & Shaver, 1987). More recently, however, individual differences in adult attachment representations have been conceptualised in terms of two dimensions: attachment anxiety and attachment avoidance (Bartholomew, 1990; Griffin & Bartholomew, 1994). Those with high levels of attachment anxiety perceive themselves to be unworthy of love and fear abandonment. Those with high levels of attachment avoidance, in contrast, tend to have negative beliefs about others and dismiss the importance of close relationships.

A number of studies have reported associations between high levels of attachment anxiety or attachment avoidance and the presence of mental health problems (e.g., Mickelson et al., 1997), including psychosis (Berry et al., 2007). There also appears to be an association between attachment style and the non-clinical psychosis phenotype, with several studies reporting associations between insecure attachment style and schizotypy (Meins et al., 2008; Tiliopolous & Goodall, 2009), and between attachment style and specific types of PLEs. High levels of both attachment anxiety and attachment avoidance are associated with high levels of paranoid thinking (Berry et al., 2006; MacBeth et al., 2008; Pickering et al., 2008) and high levels of hallucination-proneness (Berry et al., 2006; MacBeth et al., 2008). The association between attachment style and hallucination-proneness, however, appears to be a result of the association between attachment style and paranoid thinking. Pickering et al. (2008) demonstrated that both attachment anxiety and attachment avoidance failed to predict hallucination-proneness after controlling for co-occurring paranoia, which previous studies had failed to do, suggesting that the association between attachment style and hallucination-proneness is explained by the association between attachment style and paranoid thinking.

Pickering et al. (2008) also identified a number of mediators of the association between attachment style and paranoid thinking. They reported that low self-esteem, the anticipation of threat, and a belief that others have control over one's life partially mediated the association between paranoid thinking and both attachment anxiety and attachment avoidance. Another potential mediator of the association between attachment style and paranoid thinking is loneliness. A number of studies have reported associations between loneliness and low levels of attachment security (DiTommaso, Brannen-McNulty, Ross, & Burgess, 2003; Kerns, Klepac, & Cole, 1996; Larose & Bernier, 2001). In addition, several studies have reported associations between loneliness and a diagnosis of psychosis (Meltzer et al., 2013), between loneliness and the onset of psychotic experiences (Myin-Germeys, Nicolson, & Delespaul, 2001; Delespaul et al., 2002), and between loneliness and non-clinical paranoid thinking (Freeman, Pugh, et al., 2008; Riggio & Kwong, 2009). Previously, such associations were seen as reflecting the negative impact of psychotic experiences on social functioning (e.g., Møller & Husby, 2000). However, recent empirical work has suggested that loneliness may play a causal role in the development of psychotic experiences (van der Werf, van Winkel, van Boxtel, & van Os, 2010). At present, it is not clear whether loneliness is associated with hallucination-proneness and paranoid thinking, or whether, like attachment style, loneliness is related to paranoid thinking only. The present study therefore set out to examine (a) the associations between loneliness and PLEs, and (b) whether loneliness mediates the associations between paranoid thinking and both attachment avoidance and attachment anxiety.

3.3 Method

3.3.1 Participants

Participants were 368 adults (272 women, 96 men), aged 18- to 45-years ($M = 20.88$, $SD = 3.87$), who responded to adverts for a study looking at 'relationships and mental health'

on social networking websites and on a university email circular system. The study advert explained that individuals who were living in the United Kingdom, who had a good understanding of English, who did not have a history of head injury or neurological problems, and were aged 18- to 45-years were eligible to take part.

3.3.2 Procedure and measures

The study was approved by a departmental ethics committee and was conducted in accordance with the principles of the Declaration of Helsinki. The study advert directed readers to a website hosting an online questionnaire consisting of six scales. Participants provided informed consent before proceeding to the questionnaire battery. Following completion of the questionnaires, participants were presented with a debrief screen that provided contact details for the study team, as well as mental health organisations that could provide support for any distressed participants.

Participants provided their age, gender, ethnicity, and reported their perceptions of their parents' income during childhood on a 5-item scale (1 = *much less than enough money to meet our needs*; 5 = *much more than enough money to meet our needs*; Teicher, Samson, Sheu, Polcari, & McGreenery, 2010). Participants then completed six questionnaires (in the following order).

Peer Attachment Style. Attachment style in peer relationships was assessed using the Relationship Questionnaire (RQ; Bartholomew & Horowitz, 1991). This measure gives descriptions of four prototypical attachment styles (secure, dismissing, preoccupied, and fearful). Participants are asked to rate how representative of their own behaviour each description is using a 7-point Likert scale (1 = *not at all like me*; 7 = *very much like me*). Attachment anxiety is computed by subtracting participants' scores on the secure and dismissing items from the sum of their scores on the fearful and preoccupied items. Attachment avoidance is computed by subtracting participants' scores on the secure and

preoccupied items from the sum of their scores on the fearful and dismissing items. Higher scores reflect higher levels of anxiety or avoidance.

Loneliness. Loneliness was assessed using the Roberts version of the UCLA Loneliness Scale (RULS; Roberts, Lewinsohn, & Seeley, 1993). This scale consists of eight statements describing aspects of loneliness (e.g., “I feel isolated from others”). Participants are asked how often they feel this way, and respond using a 4-point Likert scale (0 = *never*; 3 = *often*). Scores range from 0 to 24, with higher scores reflecting higher levels of loneliness. In this sample, the scale had acceptable levels of internal reliability ($\alpha = .84$).

Hallucination-proneness. Hallucination-proneness was assessed using a revised version of the Launay-Slade Hallucination Scale (RHS; Launay & Slade, 1981; McCarthy-Jones & Fernyhough, 2011). This 5-item version contains only items referring to auditory experiences (e.g., “I hear a voice speaking my thoughts aloud”), and has better psychometric properties than previous versions of the Launay-Slade scale (see McCarthy-Jones & Fernyhough, 2011). Participants indicate how much each item applies to them on a 5-point Likert scale (0 = *certainly does not apply*, 4 = *certainly applies*), so that scores can range from 0 to 20, with higher scores reflecting greater hallucination-proneness. In this sample, the scale had acceptable levels of internal reliability ($\alpha = .76$).

Paranoid thinking. Paranoid thinking was assessed using the persecution items from the Persecution and Deservedness Scale (PADS; Melo et al., 2009). This scale consists of ten statements, which describe aspects of paranoid thinking (e.g., “There are times when I worry that others might be plotting against me”). Participants are asked to indicate to what extent they believe each statement on a 5-point Likert scale (0 = *certainly false*; 4 = *certainly true*), so that scores can range from 0 to 40, with higher scores reflecting higher levels of paranoid thinking. In this sample, the scale had acceptable levels of internal reliability ($\alpha = .87$).

Negative affect. Negative affect was assessed using items from the Positive and Negative Affect Schedule (Watson, Clark, & Tellegen, 1988). This scale consists of ten words that describe negative affect (e.g., scared, irritable, distressed) and participants are asked to indicate to what extent they generally (i.e., on average, rather than over a specific time-frame) feel each affective descriptor on a 5-point Likert scale (1 = *very slightly or not at all*; 5 = *extremely*). Scores can range from 10 to 50, with higher scores reflecting higher levels of negative affect. This scale had acceptable levels of internal reliability ($\alpha = .89$).

Cannabis use. Cannabis use was assessed using the revised Cannabis Use Disorders Identification Test (CUDIT-R; Adamson et al., 2010). Cannabis use is not a variable of central interest in this study, but ideally should be controlled for, given the associations between cannabis use and PLEs (Kelleher & Cannon, 2011). For the present analysis, only data collected from the first item of the CUDIT-R was used. This item asks about the frequency of cannabis use over the past six months and participants respond on a 5-point Likert scale (0 = *never*, 4 = *four or more times per week*).

3.4 Results

3.4.1 Descriptive statistics and preliminary analyses

For all demographic variables, dichotomous groups were created. In terms of ethnicity, the sample was divided into those who reported being White British ($n = 302$), and those who reported belonging to another ethnic group ($n = 65$; one participant did not report ethnicity). In terms of perceived parental income, participants were divided into one group who reported that their parents' income was less than, or much less than enough to meet their needs ($n = 31$) and a second group who reported that their parents' income was enough, more than enough, or much more than enough to meet their needs ($n = 337$). In terms of cannabis use, participants were divided into those who had used cannabis in the past six months ($n = 68$) and those who had not ($n = 293$; seven participants did not report their cannabis use).

Descriptive statistics for all variables are presented in Table 3.1. To examine the associations between the demographic variables (and cannabis use) and the predictor and outcome variables, a series of *t*-tests were performed, investigating differences between the gender, ethnic, parental income, and cannabis use groups. Not all participants provided complete data. Hence, the degrees of freedom associated with the *t*-tests reported here are not uniform. Gender differences in study variables were not significant, although there was, at trend level, a difference between men and women in terms of attachment avoidance (all other $p > .10$). Men reported higher levels of attachment avoidance ($M = 0.77$, $SD = 3.83$) than did women ($M = -0.09$, $SD = 4.00$), $t(360) = 1.78$, $p = .09$, $d = 0.21$. There were differences between the two ethnicity groups in terms of negative affect (all other $p > .16$). White British participants ($M = 11.95$, $SD = 7.62$) reported lower levels of negative affect than did other ethnic groups ($M = 14.61$, $SD = 10.00$), $t(358) = 2.37$, $p = .018$, $d = 0.33$.

There were differences between the two parental income groups in terms of negative affect, paranoid thinking and, at trend level, loneliness (all other $p > .16$). Participants who perceived that their parents' income met their needs during childhood ($M = 11.94$, $SD = 7.39$) reported lower levels of negative affect than participants who perceived that their parents' income did not meet their needs ($M = 18.04$, $SD = 13.31$), $t(27.31) = 3.83$, $p < .001$, $d = 0.76$. Participants who perceived that their parents' income met their needs during childhood ($M = 13.05$, $SD = 8.46$) reported lower levels of paranoid thinking than participants who perceived that their parents' income did not meet their needs ($M = 17.30$, $SD = 9.19$), $t(359) = 2.50$, $p = .013$, $d = 0.50$. Participants who perceived that their parents' income met their needs during childhood ($M = 7.48$, $SD = 4.44$) reported lower levels of loneliness than participants who perceived that their parents' income did not meet their needs ($M = 9.07$, $SD = 4.84$), $t(359) = 1.78$, $p = .08$, $d = 0.35$.

Table 3.1 Descriptive statistics for, and correlations between, measures of attachment, loneliness, negative affect, hallucination-proneness, and paranoid thinking

Variable	Mean	SD	Minimum – maximum	2	3	4	5	6
1. Attachment anxiety	-0.83	3.96	-12 – 11	.20*	.51*	.37*	.11	.48*
2. Attachment avoidance	0.13	4.17	-10 – 11		.32*	.11	.10	.28*
3. Loneliness	7.60	4.49	0 – 21			.55*	.14	.57*
4. Negative affect	12.39	8.12	10 – 50				.22*	.50*
5. Hallucination-proneness	6.48	5.04	0 – 20					.30*
6. Paranoid thinking	13.37	8.57	0 – 40					

* $p < .003$

There were differences between the two cannabis use groups in terms of negative affect, hallucination-proneness, paranoid thinking, and, at trend level, loneliness (all other $p > .76$). Participants who had used cannabis in the past six months reported higher levels of negative affect ($M = 14.68$, $SD = 10.58$) than those who had not ($M = 11.86$, $SD = 7.35$), $t(82.62) = 2.60$, $p = .010$, $d = 0.35$. Participants who had used cannabis in the past six months ($M = 8.38$, $SD = 5.12$) reported higher levels of hallucination-proneness than those who had not ($M = 6.04$, $SD = 4.92$), $t(359) = 3.51$, $p < .001$, $d = 0.47$. Participants who had used cannabis in the past six months ($M = 15.76$, $SD = 9.06$) reported higher levels of paranoid thinking than those who had not ($M = 12.81$, $SD = 15.76$), $t(359) = 2.58$, $p = .010$, $d = 0.34$. Participants who had used cannabis in the past six months ($M = 8.41$, $SD = 4.34$) reported higher levels of loneliness than those who had not ($M = 7.41$, $SD = 4.51$), $t(359) = 1.66$, $p = .10$, $d = 0.22$.

Correlations between variables are also presented in Table 3.1. A Bonferroni correction was applied to adjust for the number of correlations performed, meaning that a significance level of $\alpha' = .003$ was employed. Not all participants provided complete data. As a result, the size of N varies from 355–362 for the correlational analyses. Higher levels of attachment anxiety, attachment avoidance, loneliness, and negative affect were all associated with higher levels of paranoid thinking. Associations between attachment anxiety, attachment avoidance, loneliness and hallucination-proneness did not reach statistical significance. Regression analysis was, therefore, performed only to identify independent predictors of paranoid thinking.

3.4.2 Regression analyses – Paranoid thinking

Linear regression was conducted to identify independent predictors of paranoid thinking. As shown in Table 3.2, age, gender, ethnicity, perceived parental income, cannabis use, negative affect, and hallucination-proneness were entered as predictors in the first block,

Table 3.2 Summary of hierarchical regression analysis for paranoid thinking

Variable	B	SE B	β
<i>Block 1</i>			
Age	-0.03	0.01	-.14**
Gender	-0.76	0.88	-.04
Ethnicity	-0.44	1.02	-.02
Parental income	1.09	1.48	.03
Cannabis use	0.97	1.00	.04
Negative affect	0.53	0.05	.50***
Hallucination-proneness	0.28	0.08	.16**
<i>Block 2</i>			
Age	-0.02	0.01	-.09
Gender	-0.75	0.81	-.04
Ethnicity	-0.34	0.94	-.02
Parental income	1.68	1.36	.05
Cannabis use	1.17	0.92	.05
Negative affect	0.37	0.05	.35***
Hallucination-proneness	0.25	0.07	.15**
Attachment anxiety	0.65	0.10	.30***
Attachment avoidance	0.33	0.09	.16***
<i>Block 3</i>			
Age	-0.02	0.01	-.08
Gender	-0.44	0.79	-.02

Ethnicity	-0.17	0.91	-.01
Parental income	1.59	1.32	.05
Cannabis use	1.03	0.89	.05
Negative affect	0.26	0.06	.24***
Hallucination-proneness	0.26	0.07	.15***
Attachment anxiety	0.46	0.10	.21***
Attachment avoidance	0.22	0.09	.11*
Loneliness	0.51	0.10	.27***

* $p < .05$, ** $p < .01$, *** $p < .001$

with the two attachment variables entered in the second block, and loneliness entered in the third block. Collinearity diagnostics for this regression were satisfactory (minimum tolerance = .54; average VIF = 1.38), and residuals appeared to be both independent and normally distributed. The initial model was significant, $F(7, 352) = 22.50$, $p < .001$, adjusted $R^2 = .30$. In this model, age, negative affect, and hallucination-proneness were independent predictors of paranoid thinking. Inclusion of the two attachment variables improved the model, $F(9, 350) = 28.61$, $p < .001$, $\Delta R^2 = .12$. In this revised model, negative affect, hallucination-proneness, attachment anxiety, and attachment avoidance were independent predictors of paranoid thinking. Inclusion of loneliness improved the model, $F(10, 349) = 30.00$, $p < .001$, $\Delta R^2 = .04$. In this revised model, negative affect, hallucination-proneness, attachment anxiety, attachment avoidance, and loneliness were independent predictors of paranoid thinking.

3.4.3 Mediation analyses

Mediation analysis was performed to examine whether loneliness mediated the associations between the two attachment variables and paranoid thinking. Mediation was assessed by examination of the indirect effects of the predictor variables using a

Table 3.3 Mediation of the association between attachment style and paranoid thinking by loneliness

	Total effect	Direct effect	Total indirect effect		
			Point estimate	BC 95% CI	
				Lower	Upper
Attachment anxiety	.65***	.46***	.18	.10	.29
Attachment avoidance	.33***	.22*	.11	.06	.20

* $p < .05$, *** $p < .001$ BC 95% CI = Bias corrected 95% confidence interval

bootstrapping sampling procedure, with 5,000 samples, conducted by INDIRECT, an SPSS macro developed by Preacher and Hayes (2008). A variable was identified as a significant mediator if the 95% confidence interval of its associated point estimate for an indirect effect did not cross zero (Preacher & Hayes, 2004). As shown in Table 3.3, the indirect effects of both attachment anxiety and attachment avoidance on paranoid thinking through loneliness were significant, showing that there was a mediating effect. However, the direct effects of both attachment variables remained significant, suggesting that loneliness partially mediated the association between attachment style and paranoid thinking.

3.5 Discussion

This study replicated associations between attachment anxiety and paranoid thinking, between attachment avoidance and paranoid thinking, and showed that these associations were partially mediated by loneliness. These findings are consistent with previous reports that attachment style is associated with paranoid thinking (e.g., MacBeth et al., 2008).

Meanwhile, the lack of an association between attachment style and hallucination-proneness is consistent with theoretical and empirical work suggesting that attachment style does not

play an important role in the development of hallucination-proneness (e.g., Bentall & Fernyhough, 2008; Pickering et al., 2008). More widely, these findings are consistent with studies that have reported high levels of insecure attachment in people diagnosed with psychosis (Berry et al., 2007) and with models that emphasize the importance of attachment style in the development of psychotic experiences (e.g., Bentall et al., 2007).

The associations between attachment anxiety and paranoid thinking, and between attachment avoidance and paranoid thinking, were only partially mediated by loneliness. This suggests that a number of additional variables must be considered to explain the relations between attachment style and paranoid thinking. For example, Pickering et al. (2008) reported that the associations between paranoid thinking and both attachment anxiety and attachment avoidance were mediated by negative beliefs about the self, by beliefs that powerful others have control over one's life, and by beliefs that negative, threatening events will occur in the future. In addition to these variables, it is possible that attachment style may be related to paranoid thinking by predicting the ways in which a person reacts to unusual experiences. Cognitive models (e.g., Freeman, 2007) propose that paranoid thinking occurs when a person experiences an anomalous percept (e.g., one's food tasting strange) and generates a threat-based explanation (e.g., "Someone is trying to poison me") for that percept. It has been suggested that individual differences in attachment anxiety predict how a person will respond to unusual experiences (Mikulincer et al., 2003), with high levels of attachment anxiety associated with a tendency to perceive unusual events to be indicators of social threat. Thus, high levels of attachment anxiety may bring about paranoid thinking by predisposing a person to generate threat-based explanations when they experience anomalous percepts. Research that examines if this is the case would be helpful.

The association between loneliness and paranoid thinking is consistent with a number of studies. For example, two other studies have reported cross-sectional associations between

loneliness and non-clinical paranoid thinking (Freeman, Pugh, et al., 2008; Riggio & Kwong, 2009). These findings are also consistent with larger, population-based studies that have reported cross-sectional (Meltzer et al., 2013) and longitudinal (van der Werf et al., 2010) associations between loneliness and psychotic symptoms, and with data from a study involving a clinical sample, which reported that delusional thoughts tend to occur in the context of feelings of loneliness (Myin-Germeys et al., 2001).

The lack of association reported here between loneliness and hallucination-proneness is inconsistent with data from a study involving a psychotic sample, where the occurrence of hallucinations was associated with feelings of loneliness (Delepaul et al., 2002). One plausible explanation for this is that, while the majority of variables that are risk factors for clinical psychotic experiences also appear to be associated with PLEs, a minority are not (e.g., paternal age, winter birth; see Kelleher & Cannon, 2011). It is therefore possible that loneliness may be associated with hallucinations in clinical, but not in non-clinical, samples.

More widely, the associations between loneliness and paranoid thinking are consistent with research implicating loneliness in the development of a range of mental health problems (Heinrich & Gullone, 2006). Previous studies have reported longitudinal associations between loneliness and symptoms of depression (Cacioppo, Hawkley, & Thisted, 2010) and cross-sectional associations between loneliness and symptoms of anxiety (Cacioppo et al., 2006), disordered eating (Coric & Murstein, 1993), and alcohol abuse (Akerlind & Hornquist, 1992). More recent research has employed a multi-dimensional approach to loneliness, by assessing loneliness in terms of a person's social (i.e., peer), family, and romantic relationships (e.g., Lasgaard, Goossens, Bramsen, Trillingsgaard, & Elklit, 2011). This research has suggested that different mental health problems are related to different forms of loneliness. For example, self-harming behaviour is related to high levels of family, but not peer, loneliness, while social phobia is related to high levels of social, but not family,

loneliness (Lasgaard et al., 2011). There is already evidence that paranoid thinking may be associated with high levels of family, but not social or romantic, loneliness (Freeman, Pugh, et al., 2008) and further examination of the associations between paranoid thinking and different forms of loneliness is required.

The present results are also consistent with studies that have reported cross-sectional associations between attachment style and loneliness (e.g., DiTommaso et al., 2003; Kerns et al., 1996; Larose & Bernier, 2001). Given that adult attachment style is associated with poor parental bonding and childhood adversity (Mickelson et al., 1997), these findings can be considered consistent with the idea that loneliness develops as a result of difficult early interpersonal relationships (e.g., Shaver & Hazan, 1987), with individuals who are exposed to negative early social experiences feeling chronically unsatisfied by their future relationships. However, longitudinal work is needed to better understand the causal relations between these variables.

The present study suffered from a number of limitations. The study relied on information from a single source and was cross-sectional. However, previous research that has employed a longitudinal design (van der Werf et al., 2010) is consistent with the interpretation of the data presented here. That being said, further research that examines the associations reported here using a longitudinal design, and that is able to measure these variables in ways that minimize the issue of shared method variance (e.g., through the use of multiple informants), is required. Finally, it has been suggested that variables that are associated with high levels of PLEs are also risk factors for clinical psychosis (Kelleher & Cannon, 2011). It would be of interest, therefore, to examine whether the associations that have been reported between attachment style and psychosis (Berry et al., 2007) are also mediated by loneliness.

Chapter 4

Study 3: Associations between attachment style, anomalous percepts, and paranoid thinking in a non-clinical sample of adults

The findings of Study 2 are consistent with suggestions that attachment anxiety and avoidance play a role in the development of paranoid thinking, and with suggestions that attachment anxiety and avoidance are unrelated to AH-proneness. The findings of Study 2 also showed that the relations between paranoid thinking and attachment anxiety and between paranoid thinking and attachment avoidance were partially mediated by loneliness. Given that loneliness only partially mediated the associations between paranoid thinking and attachment anxiety and avoidance, a number of other factors must account for the associations between paranoid thinking and attachment anxiety and avoidance. Thus, in Study 3, another way in which high levels of attachment anxiety might bring about paranoid thinking was examined. One possibility is that individuals who report high levels of attachment anxiety tend to generate threat-based explanations of unusual experiences, and so are prone to experiencing paranoid thoughts when they experience some form of anomalous percept. Study 3 examined whether this is the case.

4.1 Abstract

According to the threat anticipation model, anomalous percepts play an important role in the development of paranoid thinking. However, given that anomalous percepts are neither necessary nor sufficient to cause paranoid thoughts, a number of factors must moderate the association between anomalous percepts and paranoid thinking. The present study assessed whether attachment style moderated the association between anomalous percepts and paranoid thinking. Participants were 160 university students (aged 18-38 years), who completed a questionnaire battery during a laboratory testing session. After controlling for demographic factors, recent cannabis use, negative affect, and attachment avoidance, the frequency of anomalous percepts and levels of attachment anxiety were significant predictors of paranoid thinking. The interaction term between anomalous percepts and attachment anxiety was also significant. Participants with high levels of attachment anxiety were likely to report paranoid thinking even when they experienced few anomalous percepts, while participants with low levels of attachment anxiety were only likely to report paranoid thinking when they experienced high levels of anomalous percepts. The present findings are consistent with the threat anticipation model of the development of paranoid thinking and suggest that attachment style may help to determine the types of appraisals a person makes when confronted with anomalous percepts.

4.2 Introduction

Paranoid thoughts are beliefs held by a person that are not shared by others, are implausible, resistant to change, distressing, and that refer to harm that is occurring, or is going to occur, to him/her as a result of the intentions of a persecutor (Freeman & Garety, 2000). Such beliefs are one of the core symptoms of schizophrenia (American Psychiatric Association, 2013), and are experienced by up to 50% of people diagnosed with that disorder (Sartorius et al., 1986). Paranoid thoughts are, however, also reported by people with other types of mental health problems (e.g., by people with affective disorders; van Os et al., 1999) and are reported by approximately 10% of the general population (Verdoux & van Os, 2002).

According to the threat anticipation model (Freeman, 2007), anomalous percepts play an important role in the development of paranoid thinking. This model—which builds upon the work of Maher (1974)—proposes that paranoid thoughts are rational attempts to explain unusual experiences caused by anomalous percepts. These anomalous percepts can take many forms. For example, they can be relatively dramatic, in the case of an auditory hallucination of hearing two voices commenting on one's behaviour. Or they can be more subtle, for example a person feeling inexplicably warm or cold, or perceiving their food to taste odd. Whatever form they take, it has been argued that their presence is taken by the experiencer as indicating that something odd is happening (Maher, 1974). Freeman (2007) argues that this feeling of something strange occurring in the environment, combined with a belief that these changes are threatening, leads to paranoid thoughts (e.g., that someone is trying to poison one's food).

Support for this model comes from several studies that have reported cross-sectional associations between experiencing anomalous percepts and high levels of paranoid (or delusional) thinking. For example, many delusional patients cite anomalous percepts, such as sudden changes in arousal or hallucinatory experiences, as events that triggered their

persecutory thoughts (Buchanan et al., 1993; Freeman et al., 2004). Consistent with this clinical research are the findings of studies that have examined the correlates of non-clinical paranoid thinking. For example, Bell, Halligan, and Ellis (2006) reported a large positive association ($r = .60$) between the frequency of anomalous percepts and degree of delusional thinking. However, the value of this study for understanding the relation between anomalous percepts and paranoid thinking is limited, because the measure of delusional beliefs employed assesses a range of delusions (e.g., ideas of reference, grandiose delusions) not just persecutory delusions. Better support for the role of anomalous percepts in paranoid thinking specifically, comes from the work of Tone, Goulding, and Compton (2011), who reported that the extent to which participants experienced anomalous percepts predicted scores on four of the five aspects of paranoid thinking they examined.

Consistent with these findings are studies that have examined paranoid thinking under laboratory conditions. In one study, it was shown that inducing hearing impairments (through hypnosis) could lead to an increase in paranoid thinking following social interactions (Zimbardo, Andersen, & Kabat, 1981). Other support comes from two studies that have employed virtual reality (VR) as a way of inducing paranoid thoughts. In these studies, participants are taken on a four minute virtual journey on the London underground system, which is populated by a number of neutral characters. Following the journey, participants complete a scale assessing to what extent they experienced paranoid thinking during the VR. In the first study using the VR paradigm, a number of variables were shown to predict paranoid thinking in the VR environment, including trait levels of paranoia, depression, anxiety, negative beliefs about the self and others, loneliness, and anomalous percepts (Freeman, Pugh, et al., 2008). After controlling for associations between these predictors, anomalous percepts remained a significant predictor of paranoid thinking. In a subsequent study, it was shown that trait levels of anomalous percepts were important in distinguishing

participants who reported paranoia during the VR from those who reported social anxiety during the VR (Freeman, Gittins, et al., 2008).

Together, these studies provide support for the suggestion that anomalous percepts play an important role in the development of paranoid thinking. However, not all research is consistent with this claim. For example, Bell, Halligan, and Ellis (2008) compared levels of anomalous percepts in three groups: healthy controls, a group of delusional patients who were also experiencing hallucinations, and a group of delusional patients who had not experienced hallucinations. As would be expected, the hallucinating patient group reported very high levels of anomalous percepts. However, the non-hallucinating patient group and the healthy control group reported similar levels of anomalous percepts. Bell et al. (2008) therefore concluded that high levels of anomalous percepts are not necessary for the development of paranoid or delusional thinking.

One way of explaining Bell et al.'s (2008) results is that one could argue that even low levels of anomalous percepts may lead to the development of paranoid thinking in people who are predisposed to generate threat-based explanations of their experiences. One factor that may predispose a person to generate threat-based accounts is attachment style, in particular high levels of attachment anxiety. A number of studies have reported that high levels of attachment anxiety are associated with paranoid thinking (MacBeth et al., 2008; Meins et al., 2008; Pickering et al., 2008), and this tendency to generate threat-based explanations may be one way in which attachment anxiety leads to paranoid thoughts. According to Mikulincer et al. (2003), high levels of attachment anxiety are associated with a set of emotion regulation strategies that “result in a tendency to detect threats in nearly every transaction with the physical and social world and to exaggerate the potential negative consequences of these threats” (p. 85). This account suggests that high levels of attachment anxiety might encourage a person to interpret an anomalous percept as an indicator of social

threat, and so experience paranoid thoughts. If this is the case, attachment anxiety should moderate the association between anomalous percepts and paranoid thinking (i.e., in a regression analysis, the anomalous percepts \times attachment anxiety interaction term should predict variance in paranoid thinking). High levels of attachment avoidance are also associated with high levels of paranoid thinking (e.g., Macbeth et al., 2008; Meins et al., 2008; Pickering et al., 2008). However, attachment avoidance is thought to be associated with a set of emotion regulation strategies that involve the dismissal or suppression of threat-related cues (Shaver & Mikulincer, 2002). Thus, unlike attachment anxiety, attachment avoidance should not moderate the association between anomalous percepts and paranoid thinking. To examine whether this was the case, we investigated associations between attachment style, anomalous percepts, and paranoid thinking in a non-clinical sample.

4.3 Method

4.3.1 Participants

Participants were 160 university students (137 women) aged 18- to 38-years ($M = 21.08$, $SD = 3.44$) who took part in exchange for course credit or a small payment.

Participants had a good understanding of English, did not have a history of head injury or neurological problems, and did not have any vision or hearing problems.

4.3.2 Procedure and measures

The study was approved by a departmental ethics committee and was conducted in accordance with the principles of the Declaration of Helsinki. Participants were tested in a quiet laboratory. After providing informed consent, participants completed a questionnaire battery, which included the measures below. The order in which participants completed these measures and tasks was counter-balanced, so that half of the sample completed them in the order presented below, and half completed them in the reverse order. Task order had no effect on any of the variables included in the analysis.

Demographics. Participants provided their age, gender, ethnicity, and reported their perceptions of their parents' income during childhood on a 5-item scale (1 = *much less than enough money to meet our needs*; 5 = *much more than enough money to meet our needs*; Teicher et al., 2010).

Peer Attachment Style. Attachment style in peer relationships was assessed using the Relationship Questionnaire (RQ; Bartholomew & Horowitz, 1991). This measure gives descriptions of four prototypical attachment styles (secure, dismissing, preoccupied, and fearful). Participants are asked to rate how representative of their own behaviour each description is on a 7-point Likert scale (1 = *not at all like me*; 7 = *very much like me*). Attachment anxiety is found by subtracting participants' scores on the secure and dismissing items from the sum of their scores on the fearful and preoccupied items. Attachment avoidance is found by subtracting participants' scores on the secure and preoccupied items from the sum of their scores on the fearful and dismissing items. Higher scores reflect higher levels of anxiety or avoidance.

Anomalous Percepts. Frequency of anomalous percepts was assessed using the Cardiff Anomalous Perceptions Scale (CAPS; Bell et al., 2006). This is a 32-item scale, with each item describing a different type of anomalous percept (e.g., "Do you ever notice that food or drink seems to have an unusual taste?"). The original CAPS asked respondents to indicate whether or not they have experienced the anomalous percept described in each item, and, if they responded yes, to rate how frequent, how distressing, and how intrusive this percept was. The present study, however, was only concerned with the frequency of anomalous percepts. Respondents were therefore only asked to rate how often they experienced each percept on a 6-point Likert scale (0 = *never*; 5 = *happens all the time*). Potential scores ranged from 0 to 160, with higher scores reflecting more frequent anomalous percepts. In this sample, the scale had acceptable levels of internal reliability ($\alpha = .93$).

Paranoid Thinking. Paranoid thinking was assessed using the persecution items from the Persecution and Deservedness Scale (PADS; Melo et al., 2009). This scale consists of ten statements, which describe aspects of paranoid thinking (e.g., “There are times when I worry that others might be plotting against me”). Participants are asked to indicate to what extent they believe each statement on a 5-point Likert scale (0 = *certainly false*; 4 = *certainly true*), so that scores can range from 0 to 40, with higher scores reflecting higher levels of paranoid thinking. In this sample, the scale had acceptable levels of internal reliability ($\alpha = .87$).

Negative Affect. Negative affect was assessed using items from the Positive and Negative Affect Schedule (PANAS; Watson et al., 1988). This scale consists of ten words that describe negative affect (e.g., scared, irritable, distressed) and participants are asked to indicate to what extent they generally (i.e., on average, rather than over a specific time-frame) feel each affective descriptor on a 5-point Likert scale (1=*very slightly or not at all*; 5=*extremely*). Scores can from 10 to 50, with higher scores reflecting higher levels of negative affect. This scale had acceptable levels of internal reliability ($\alpha = .88$).

Cannabis use. Cannabis use was assessed using the revised Cannabis Use Disorders Identification Test (CUDIT-R; Adamson et al., 2010). Cannabis use is not a variable of central interest in this study, but ideally should be controlled for, given the associations between cannabis use and non-clinical paranoid thinking (Kelleher & Cannon, 2011). For the present analysis, only data collected from the first item of the CUDIT-R was used. This item asks about the frequency of cannabis use over the past six months and participants respond on a 5-point Likert scale (0 = *never*, 4 = *four or more times per week*).

4.4 Results

4.4.1 Descriptive statistics and preliminary analyses

For all demographic variables, dichotomous groups were created. In terms of ethnicity, the sample was divided into those who reported being White British ($n = 120$), and

those who reported belonging to another ethnic group ($n = 39$; one participant did not report ethnicity, nor any other demographic information). In terms of perceived parental income, the sample was divided into one group who reported that their parents' income was less than, or much less than enough to meet their needs ($n = 18$) and a second group who reported that their parents' income was enough, more than enough, or much more than enough to meet their needs ($n = 141$). In terms of cannabis use, participants were divided into those who had used cannabis in the past six months ($n = 28$) and those who had not ($n = 131$).

Descriptive statistics for all variables are presented in Table 4.1. To examine the associations between the demographic variables (and cannabis use) and the predictor and outcome variables, a series of t -tests were performed, investigating differences between the gender, ethnic, parental income, and cannabis use groups. Gender differences in study variables were not significant (all $p > .16$). Differences between the two ethnicity groups for all study variables were not significant (all $p > .40$), except, at the trend level, in terms of frequency of anomalous percepts. White British participants reported slightly less frequent anomalous percepts ($M = 24.44$, $SD = 19.80$) than did other ethnic groups ($M = 31.54$, $SD = 21.81$), $t(157) = 1.90$, $p = .06$, $d = 0.35$.

Differences between the two parental income groups for all study variables were not significant (all $p > .10$), except, at the trend level, in terms of paranoid thinking. Participants who perceived that their parents' income met their needs during childhood ($M = 11.47$, $SD = 7.99$) reported lower levels of paranoid thinking than participants who perceived that their parents' income did not meet their needs ($M = 15.33$, $SD = 9.18$), $t(157) = 1.90$, $p = .06$, $d = 0.47$.

There were differences between the two cannabis use groups in terms of negative affect and, at the trend level, in terms of paranoid thinking (all other $p > .29$). Participants who had used cannabis in the past six months ($M = 22.36$, $SD = 7.04$) reported higher levels.

Table 4.1 Descriptive statistics for, and correlations between, measures of attachment, anomalous percepts, negative affect, and paranoid thinking

Variable	Mean	SD	Minimum – maximum	2	3	4	5
1. Attachment anxiety	-0.78	4.12	-12 – 11	.12	.24*	.39*	.47*
2. Attachment avoidance	-0.47	4.07	-9 – 10		.04	.18	.30*
3. Anomalous percepts	26.19	20.41	0 – 124			.40*	.49*
4. Negative affect	20.03	6.83	10 – 40				.60*
5. Paranoid thinking	12.02	8.29	0 – 36				

* $p < .005$

of negative affect than did participants who had not used cannabis in the past six months ($M = 19.42$, $SD = 6.62$), $t(155) = 2.11$, $p = .037$, $d = 0.43$. Participants who had used cannabis in the past six months ($M = 14.54$, $SD = 9.47$) reported higher levels of paranoid thinking than did participants who had not used cannabis in the past six months ($M = 11.35$, $SD = 7.82$), $t(157) = 1.89$, $p = .06$, $d = 0.39$.

Correlations between variables are also presented in Table 4.1. A Bonferroni correction was applied to adjust for the number of correlations performed, meaning that a significance level of $\alpha' = .005$ was employed. Not all participants provided complete data. As a result, the size of N varies from 158–160 for the correlational analyses. Higher levels of attachment anxiety, attachment avoidance, anomalous percepts, and negative affect were all associated with higher levels of paranoid thinking.

4.4.2 Regression and moderation analyses

Linear regression analysis was conducted to identify independent predictors of paranoid thinking and to examine whether attachment anxiety moderated the association

Table 4.2 Summary of hierarchical regression analysis for paranoid thinking

Variable	B	SE B	β
<i>Block 1</i>			
Age	0.02	0.01	.10
Gender	0.34	1.59	.02
Ethnicity	-1.31	1.31	-.07
Parental income	3.71	1.69	.15*
Recent cannabis use	1.04	1.42	.05
Negative affect	0.69	0.08	.57***
<i>Block 2</i>			
Age	0.02	0.01	.08
Gender	-0.31	1.38	-.01
Ethnicity	-1.61	1.13	-.08
Parental income	4.25	1.49	.17**
Recent cannabis use	1.89	1.23	.09
Negative affect	0.38	0.08	.31***
Anomalous percepts	0.14	0.03	.35***
Attachment anxiety	0.41	0.13	.20**
Attachment avoidance	0.36	0.12	.18**
<i>Block 3</i>			
Age	0.01	0.01	.05
Gender	-0.5	1.37	-.03
Ethnicity	-1.67	1.12	-.09

Parental income	4.41	1.47	.17**
Recent cannabis use	1.78	1.21	.08
Negative affect	0.40	0.08	.33***
Anomalous percepts	0.14	0.03	.36***
Attachment anxiety	0.46	0.13	.23***
Attachment avoidance	0.34	0.12	.17**
Anomalous percepts × attachment anxiety	-0.01	0.01	-.14*
Anomalous percepts × attachment avoidance	0.00	0.01	.04

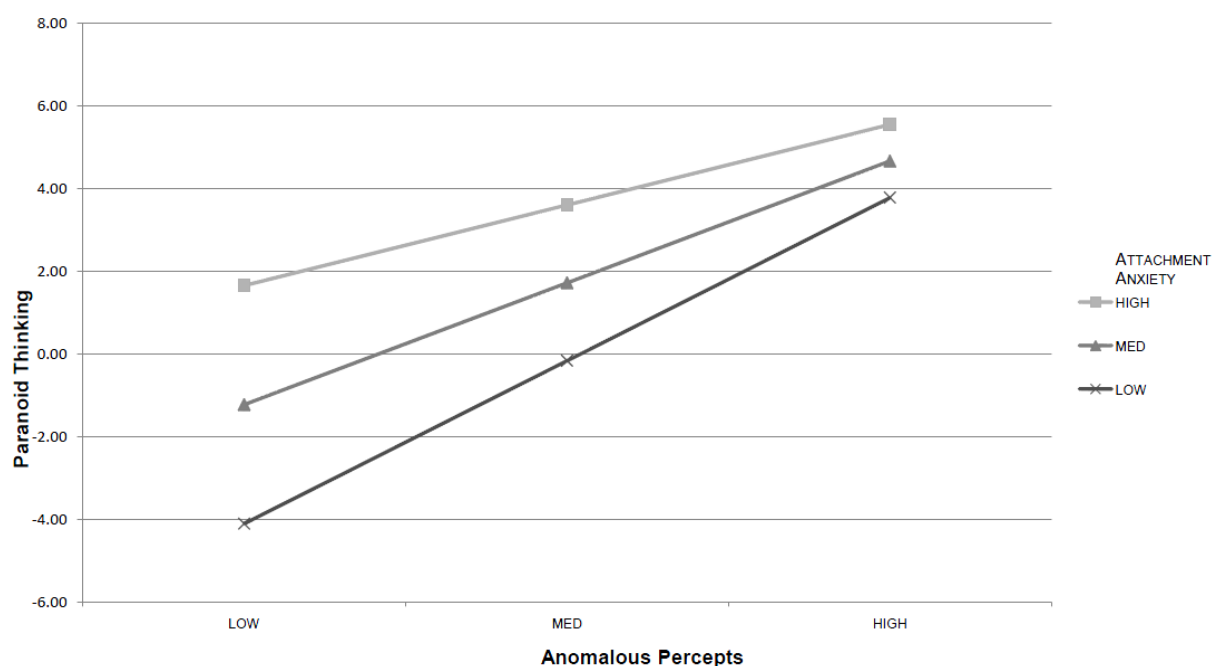
* $p < .05$, ** $p < .01$, *** $p < .001$

between anomalous percepts and paranoid thinking. As shown in Table 4.2, age, gender, ethnicity, perceived parental income, recent cannabis use, and negative affect were entered as predictors in the first block. Frequency of anomalous percepts and the two attachment variables were entered in the second block. Following Aiken and West (1991), frequency of anomalous percepts, attachment anxiety, and attachment avoidance were centred prior to being entered into the regression analyses. The interaction terms anomalous percepts × attachment anxiety and anomalous percepts × attachment avoidance, calculated by multiplying the relevant centred variables, were entered in the third block. Collinearity diagnostics for this regression were satisfactory (minimum tolerance = .68; average VIF = 1.21), and residuals appeared to be both independent and normally distributed. The initial model was significant, $F(6, 148) = 15.13, p < .001$, adjusted $R^2 = .36$. In this model, parental income and negative affect were significant predictors of paranoid thinking. Inclusion of frequency of anomalous percepts and the two attachment variables improved the model, $F(9, 145) = 20.29, p < .001, \Delta R^2 = .15$. In this model, parental income, negative affect, anomalous

percepts, attachment anxiety, and attachment avoidance were significant predictors of paranoid thinking. Inclusion of the two interactions terms improved the model, $F(11, 143) = 17.54, p < .001, \Delta R^2 = .02$. In this model, parental income, negative affect, anomalous percepts, attachment anxiety, attachment avoidance, and the anomalous percepts \times attachment anxiety interaction term were significant predictors of paranoid thinking.

To interpret the anomalous percepts \times attachment anxiety interaction effect, ModGraph (Jose, 2008)—a programme that graphically displays interaction effects—was employed. As shown in Figure 4.1, when the frequency of anomalous percepts was high, low levels of attachment anxiety did not protect participants from experiencing high levels of paranoid thinking. However, when the frequency of anomalous percepts was medium or low, participants who reported low and medium levels of attachment anxiety experienced lower levels of paranoid thinking than participants who reported high levels of attachment anxiety.

Figure 4.1 Moderation of the association between anomalous percepts and paranoid thinking by attachment anxiety.



4.5 Discussion

The present results suggest that high levels of attachment avoidance, high levels of attachment anxiety, and frequent anomalous percepts are associated with high levels of paranoid thinking. Importantly, the present findings also suggest that these latter two factors interact, albeit in a manner different to our predictions. When participants experienced relatively infrequent anomalous percepts, individual differences in attachment anxiety were important in predicting which participants were likely to report low levels of paranoid thinking (those who reported low levels of attachment anxiety), and which participants were likely to report high levels of paranoid thinking (those who reported high levels of attachment anxiety). However, when participants experienced frequent anomalous percepts, individual differences in attachment anxiety were not important in predicting which participants were likely to report high levels of paranoid thinking.

Primarily, these findings are consistent with previous reports of associations between self-reported levels of anomalous percepts and paranoid thinking in non-clinical samples (Bell et al., 2006; Freeman, Gittins, et al., 2008; Freeman, Pugh, et al., 2008; Tone et al., 2011), and with research that has shown that experimentally-induced anomalous percepts can elicit paranoid thinking in healthy young adults (Zimbardo et al., 1981). These analogue studies are concordant with clinical studies that have shown that paranoid or deluded patients often cite internal experiences as evidence that supports their unusual beliefs (Buchanan et al., 1993; Freeman et al., 2004). Together, these findings are consistent with Freeman's (2007) threat anticipation model and Maher's (1974) work, which both propose that anomalous percepts play an important role in the development of paranoid thinking.

Importantly, the present results may account for Bell et al.'s (2008) finding that healthy control participants reported similar levels of anomalous percepts to deluded, non-hallucinating participants. Bell et al. wrote that their findings indicated that anomalous

percepts do not have to be present for paranoid thinking to develop, as some delusional patients appear to experience low levels of anomalous percepts. The present results are concordant with that view, and suggest that paranoid thinking can develop in the context of high levels of attachment anxiety, even in the absence of anomalous percepts. A number of studies have shown that people diagnosed with schizophrenia report high levels of attachment anxiety (e.g., Mickelson et al., 1997). Thus, it is plausible that Bell et al.'s deluded, non-hallucinating participants would have reported high levels of attachment anxiety had this variable been assessed in that study. Research that examines how anomalous percepts and attachment anxiety interact with one another to predict paranoid thinking in a clinical sample would be helpful.

The present findings also replicate the associations between attachment style and non-clinical paranoid thinking that have been reported elsewhere (MacBeth et al., 2008; Meins et al., 2008; Pickering et al., 2008). More broadly, these findings are consistent with reports of high levels of insecure attachment in psychotic patients (Ponizovsky et al., 2007, 2013), with reports that changes in attachment security over time predict changes in psychotic symptoms (Berry et al., 2008), and with approaches that posit an important role for attachment in the development of paranoid thinking (e.g., Bentall & Fernyhough, 2008; Bentall et al., 2007).

The moderating role of attachment anxiety reported here is consistent with Mikulincer et al.'s (2003) suggestion that high levels of attachment anxiety are associated with a tendency to exaggerate the presence of social threat. This has been demonstrated by Meyer, Olivier, and Roth (2005), who showed that participants who reported high levels of attachment anxiety rated hypothetical scenarios (e.g., your romantic partner cancels plans with you so that they can study with a friend) as more threatening to their relationship than did participants who report low levels of attachment anxiety. The body of evidence demonstrating an association between appraisals of social threat and levels of attachment

anxiety is, however, relatively small, and additional research examining the strength of this association is required.

The finding that attachment avoidance failed to moderate the association between anomalous percepts and paranoid thinking was in line with our predictions and is consistent with Shaver and Mikulincer's (2002) conception of the emotion regulation strategies associated with attachment avoidance. These strategies are characterised by attempts to deactivate distress, so rather than interpreting anomalous percepts as signs of social threat, one might expect participants who report high levels of attachment avoidance to dismiss the importance of anomalous percepts, or to attempt to suppress them. While a number of variables that mediate the associations between attachment avoidance and paranoid thinking have been identified (e.g., Pickering et al., 2008), further research is required to identify the mechanisms by which high levels of attachment avoidance lead to paranoid thinking. The theoretical framework provided by Shaver and Mikulincer (2002) should prove to be a useful basis for this work. For example, participants who engage in thought suppression, one of the deactivating strategies described by Shaver and Mikulincer, experience more frequent intrusive thoughts than do participants who do not engage in thought suppression (Jones & Fernyhough, 2006, 2009; Salkovskis & Campbell, 1994). Intrusive thoughts may play a role in the development of paranoid thinking (Varese, Barkus, & Bentall, 2011), and so it would be interesting to examine whether the tendency to employ thought suppression mediates the association between attachment avoidance and paranoid thinking.

The present study suffered from a number of limitations. The study relied on information from a single source and was cross-sectional, meaning that some of the associations reported here may be inflated due to shared method variance and that no conclusions can be drawn about the causal relations we have proposed here. However, previous studies that have employed an experimental design to examine the associations

between anomalous percepts and paranoid thinking (e.g., Zimbardo et al., 1981), or a longitudinal design to examine associations between attachment style and psychotic experiences (Berry et al., 2008), are consistent with the interpretation of the data presented here. That said, further research that examines the associations reported here, employs a longitudinal design, and that avoids the issue of shared method variance is required.

Chapter 5

Study 4: Associations between intrusive thoughts, reality discrimination, and auditory hallucination-proneness in a non-clinical sample of adults

The studies reported in Chapters 5 and 6 addressed research questions related to a cognitive bias which is thought to be important in the development of auditory hallucinations (AH), but not in the development of paranoid thinking. Reality discrimination refers to a person's ability to distinguish internal, self-generated events from external, non-self-generated events. People who experience AH, or who are prone to AH, show biased reality discrimination, so that they frequently misattribute internal, self-generated events to an external, non-self source, but rarely make the reverse error. A number of researchers have proposed that reality discrimination biases should interact with other cognitive factors, such as the presence of intrusive cognitions, to predispose a person to experiencing AH. However, these claims have not yet been examined empirically. Thus, the aim of Study 5 was to examine associations between reality discrimination biases, intrusive thoughts, and AH-proneness, and to test whether reality discrimination biases moderate the association between intrusive thoughts and AH-proneness.

5.1 Abstract

People who experience intrusive thoughts are at increased risk of developing hallucinatory experiences, as are people who have weak reality discrimination skills. No study has yet examined whether these two factors interact to make a person especially prone to hallucinatory experiences. The present study examined this question in a non-clinical sample. Participants were 160 students, who completed a reality discrimination task, as well as self-report measures of cannabis use, negative affect, intrusive thoughts, and auditory hallucination-proneness. The possibility of an interaction between reality discrimination performance and level of intrusive thoughts was assessed using multiple regression. The number of reality discrimination errors participants made and the level of intrusive thoughts reported were independent predictors of hallucination-proneness. The reality discrimination errors \times intrusive thoughts interaction term was significant, so that participants who made many reality discrimination errors and reported high levels of intrusive thoughts were especially prone to hallucinatory experiences. If applicable to clinical samples, these findings suggest that reducing the number of intrusive thoughts patients experience may reduce the frequency of hallucinatory experiences. They also suggest that, if possible, improving a person's reality discrimination skills may prove to be another way in which the frequency of hallucinatory experiences could be reduced.

5.2 Introduction

Cognitive models of auditory hallucinations (AH) typically agree that they occur when an internal, mental event is misattributed to an external source (e.g., Bentall, 1990; Frith, 1992; Hoffman, 1986; Waters et al., 2012). However, there is little agreement over what types of mental event are the “raw cognitive material from which hallucinations are constructed” (Elua, Laws, & Kvavilashvili, 2012, p. 166). It has been suggested (e.g., Morrison, Haddock, & Tarrier, 1995) that intrusive thoughts may be this raw cognitive material. This is, in part, because intrusive thoughts and AH share a number of important features: both have been described as being unwanted, uncontrollable, and distressing (Rachman, 1978, 1981; Nayani & David, 1996). Morrison (Morrison et al., 1995; Morrison, 2001) has offered a meta-cognitive account of how intrusive thoughts might be experienced as externally-generated, which proposed that intrusive thoughts that are ego-dystonic might be attributed to an external source so as to reduce cognitive dissonance. There is little evidence to support this ‘dissonance reduction’ account (see Varese & Bentall, 2011), but there is some evidence to support a role for intrusive thoughts in the development of AH. For example, studies with clinical populations have reported that voice-hearers experience more intrusive thoughts than do psychiatric controls (Lobban, Haddock, Kinderman, & Wells, 2002; Morrison & Baker, 2000). Meanwhile, analogue studies have shown that high levels of intrusive thoughts are associated with AH-proneness in non-clinical participants (Jones & Fernyhough, 2006, 2009). Together, these findings suggest that while there is a lack of support for some aspects of Morrison’s meta-cognitive account, intrusive thoughts may prove to be the raw material from which AH are formed.

Studies examining source memory, which tend to involve participants trying to recall whether they or the experimenter generated a set of items, have suggested one explanation for why intrusive thoughts may play this role in the development of AH. In these studies (e.g.,

Brébion et al., 2000), participants are presented with a set of cues, such as a question or the first part of a word-pair, and they or the experimenter must generate a word in response to each cue. At test, participants are presented with the responses they generated, as well as the responses generated by the experimenter, and must recall who generated each item. A number of studies have shown that self-generated items that require little cognitive effort to generate are more likely to be misattributed to an external source than are items which required considerable cognitive effort to generate (see Johnson, Hashtroudi, & Lindsay, 1993). For example, in one trial a participant may be asked to name a fruit beginning with the letter 'A'. Participants tend to answer 'apple' and generating that answer normally requires little cognitive effort. In a subsequent trial that participant may be asked to name a fruit beginning with the letter 'T'. The participant might generate the answer 'tomato' and this answer tends to require more cognitive effort than the answer 'apple'. At test, participants are less likely to recall generating 'apple' than 'tomato'. According to Johnson's (e.g., 1997) Source Monitoring Framework, this is because the effort of generating the latter answer has left a set of cognitive cues that help participants to identify themselves as the source of the word. These cognitive cues are less likely to exist for low-effort answers and so these are more likely to be misattributed to an external source. It has been argued (e.g., Bentall, 2003) that the unbidden nature of intrusive thoughts means that they tend to lack various features that suggest to a person that they were the author of that thought (i.e., they are not associated with any cognitive effort). For this reason, they are more likely to be attributed to an external source and this may explain the associations between intrusive thoughts and hallucinatory experiences.

Another factor that appears to play a role in the development of AH is reality discrimination. Reality discrimination refers to the process by which a person distinguishes between internal, self-generated and external, other-generated events (Bentall, 1990). One

way of assessing reality discrimination is through an auditory signal detection task (SDT). In this task, participants must try to detect a signal (typically a small amount of speech) in an ambiguous auditory stimulus (typically white noise). On some trials the signal is present, on other trials the signal is absent. Reality discrimination errors occur when a participant makes a false alarm—that is, when they perceive speech to be present in the white noise when it is absent. Presumably, when a false alarm occurs, participants have mistaken their internal representation of the signal (the speech) for the external signal. A recent meta-analysis (Brookwell et al., 2013) has shown that psychotic participants who experience AH, and participants from a non-clinical population who are prone to hallucinatory experiences, make more false alarms than do control participants when performing these tasks, suggesting that they have a tendency to misattribute internally-generated events to an external source.

Bentall (2003) has argued that a person who experiences high levels of intrusive thoughts *and* displays poor reality discrimination abilities should be especially prone to AH, as they will frequently experience cognitions that lack the characteristics of self-generated events and be predisposed towards mistaking internal, self-generated events for external, other-generated events. No study has yet examined whether this is the case. Therefore, in the present study we set out to examine this question by investigating, in an analogue sample, whether a combination of poor reality discrimination skills and intrusive thoughts placed a person at an especially high risk of hallucinatory experiences.

5.3 Method

5.3.1 Participants

Participants were 160 university students (137 women), aged 18- to 38-years ($M = 21.08$, $SD = 3.44$), who received course credit or a small payment in return for their time. Participants had a good understanding of English, did not have a history of head injury or neurological problems, and did not have any hearing problems.

5.3.2 Procedure and measures

The study was approved by a departmental ethics committee and was conducted in accordance with the principles of the Declaration of Helsinki. Participants were tested in a quiet laboratory. After providing informed consent, participants completed the signal detection task and a questionnaire battery, which included the measures below. The order in which participants completed these measures and tasks was counter-balanced, so that half of the sample completed them in the order presented below, and half completed them in the reverse order. Task order had no effect on any of the variables included in the analysis.

Demographics. Participants provided their age, gender, ethnicity, and reported their perceptions of their parents' income during childhood on a 5-item scale (1 = *much less than enough money to meet our needs*; 5 = *much more than enough money to meet our needs*; Teicher et al., 2010).

Cannabis use. Cannabis use was assessed using the revised Cannabis Use Disorders Identification Test (CUDIT-R; Adamson et al., 2010). Cannabis use is not a variable of central interest in this study, but ideally should be controlled for, given the associations between cannabis use and non-clinical AH (Kelleher & Cannon, 2011). For the present analysis, only data collected from the first item of the CUDIT-R was used. This item asks about the frequency of cannabis use over the past six months and participants respond on a 5-point Likert scale (0 = *never*, 4 = *four or more times per week*).

Intrusive thoughts. The level of intrusive thoughts experienced by participants was assessed using the intrusions subscale of the White Bear Suppression Inventory (WBSI; Wegner & Zanakos, 1994). The WBSI is a 15-item scale and five of these items assess to what extent participants experience intrusive thoughts (Muris, Merckelbach, & Horselenberg, 1996). These five items describe various aspects of thought intrusions (e.g., "I have thoughts I cannot stop") and participants indicate to what extent they agree with each statement on a 5-

point Likert scale (1 = *strongly disagree*; 5 = *strongly agree*), so that scores can range from 5–25, with higher scores reflecting a higher level of intrusive thoughts. In this sample, the scale had acceptable levels of internal reliability ($\alpha = .84$).

Auditory hallucination-proneness. AH-proneness was assessed using the nine items from the Cardiff Anomalous Perceptions Scale (CAPS; Bell et al., 2006) that assess unusual auditory experiences. These nine items ask whether participants have experienced various unusual auditory percepts (e.g., “Do you ever hear noises or sounds when there is nothing around to explain them?”). Participants rate how often they have had these experiences on a 6-point Likert scale (0 = *never*; 5 = *happens all the time*), so that scores can range from 0–45, with higher scores reflecting greater AH-proneness. The original CAPS includes items that assess anomalous percepts in other sensory domains (e.g., visual, tactile, and olfactory), and asks about how distressing and intrusive these percepts are. In the present study, however, we are only concerned with AH-proneness, and with the frequency of these experiences. In this sample, the scale had acceptable levels of internal reliability ($\alpha = .81$).

Negative affect. Negative affect was assessed using items from the Positive and Negative Affect Schedule (Watson et al., 1988). This scale consists of ten words that describe negative affect (e.g., scared, irritable, distressed) and participants are asked to indicate to what extent they generally (i.e., on average, rather than over a specific time-frame) feel each affective descriptor on a 5-point Likert scale (1 = *very slightly or not at all*; 5 = *extremely*). Scores can range from 10 to 50, with higher scores reflecting higher levels of negative affect. This scale had acceptable levels of internal reliability ($\alpha = .88$).

Auditory signal detection task. To assess reality discrimination, participants completed the auditory SDT described in Barkus et al. (2007). This task consisted of 60 trials, with each trial consisting of a five second burst of white noise followed by three seconds of silence. In 34 of the bursts of white noise, one second of speech was presented. In the

remaining 26 bursts of white noise, no speech was presented. In 12 of the trials when speech was presented, the speech was clearly audible. In the remaining 22 trials, the speech was presented at an auditory threshold. This threshold was determined prior to the start of testing by establishing the volume of speech that was perceived by 50% of a small sample ($n = 10$) of participants who were around the same age as the experimental participants. The task was presented to participants on a laptop computer, using the audio recording and playing software package Cakewalk (www.cakewalk.com), and participants listened to the task stimuli using standard Sony headphones.

Participants were asked to decide whether or not speech had been presented in each burst of white noise, and to respond via a button press during the three seconds of silence that followed the white noise. The number of false alarms (i.e., trials where participants had responded that the speech was present in the white noise when it was, in fact, absent) participants made was recorded. False alarms were used as a measure of reality discrimination, with a larger number of false alarms indicating weaker reality discrimination. While many studies that employ the SDT to assess reality discrimination abilities use β —a measure of participants' response bias—as their main outcome (e.g., Varese et al., 2011), several use the number of false alarms as their main outcome (e.g., Barkus et al., 2011).

5.4 Results

5.4.1 Descriptive statistics and preliminary analyses

For all demographic variables, dichotomous groups were created. In terms of ethnicity, the sample was divided into those who reported being White British ($n = 120$), and those who reported belonging to another ethnic group ($n = 39$; one participant did not report ethnicity, or any other demographic information). In terms of perceived parental income, participants were divided into one group who reported that their parents' income was less than, or much less than enough to meet their needs ($n = 18$) and a second group who reported

that their parents' income was enough, more than enough, or much more than enough to meet their needs ($n = 141$). In terms of cannabis use, participants were divided into those who had used cannabis in the past six months ($n = 28$) and those who had not ($n = 131$).

Descriptive statistics for all variables are presented in Table 5.1. To examine the associations between the demographic variables (and cannabis use) and the predictor and outcome variables, a series of t -tests were performed, investigating differences between the gender, ethnic, parental income, and cannabis use groups. Gender differences in negative affect, intrusive thoughts, number of false alarms, and AH-proneness were not significant (all $p > .57$). There was a difference between the two ethnicity groups, at trend level, in terms of level of intrusive thoughts (all other $p > .18$). White British participants ($M = 15.76$, $SD = 4.75$) reported lower levels of intrusive thoughts than did other ethnic groups ($M = 17.29$, $SD = 3.91$), $t(156) = 1.80$, $p = .07$, $d = 0.33$.

There were differences between the two parental income groups in terms of the number of false alarms made (all other $p > .26$). Participants who perceived that their parents' income met their needs during childhood ($M = 4.65$, $SD = 4.45$) made more false alarms than did participants who perceived that their parents' income did not meet their needs ($M = 2.06$, $SD = 1.98$), $t(151) = 2.36$, $p = .019$, $d = 0.59$. There were differences between the two cannabis use groups in terms of negative affect (all other $p > .67$). Participants who had used cannabis in the past six months ($M = 22.36$, $SD = 7.04$) reported higher levels of negative affect than did participants who had not used cannabis in the past six months ($M = 19.41$, $SD = 6.59$), $t(156) = 2.12$, $p = .037$, $d = 0.43$.

5.4.2 Correlational and multiple regression analyses

Correlations between variables are also presented in Table 5.1. A Bonferroni correction was applied to adjust for the number of correlations performed, meaning that a

Table 5.1 Descriptive statistics for, and correlations between, measures of negative affect, intrusive thoughts, number of false alarms, and auditory hallucination-proneness

Variable	Mean	SD	Minimum – maximum	2	3	4
1. Negative affect	20.02	6.81	10 – 40	.45*	.22*	.41*
2. Intrusive thoughts	16.15	4.59	5 – 25		.19	.54*
3. False alarms	4.36	4.32	0 – 25			.38*
4. Auditory hallucination-proneness	7.02	6.21	0 – 32			

* $p < .008$

significance level of $\alpha' = .008$ was employed. Not all participants provided complete data. As a result, the size of N varies from 153 to 159 for the correlational analyses. Higher levels of negative affect, intrusive thoughts, and more false alarms were all associated with higher levels of AH-proneness.

A linear regression was conducted to identify independent predictors of AH-proneness. As shown in Table 5.2, age, gender, ethnicity, perceived parental income, cannabis use, and negative affect were entered as predictors in the first block. In the second block, intrusive thoughts and number of false alarms were entered, with the intrusive thoughts \times number of false alarms interaction term entered in the third block. Collinearity diagnostics for this regression were satisfactory (minimum tolerance = .72; average VIF = 1.22), and residuals appeared to be both independent and normally distributed. The initial model was significant, $F(6, 146) = 5.45, p < .001$, adjusted $R^2 = .15$. In this model, only negative affect was an independent predictor of AH-proneness. Inclusion of intrusive thoughts and number of false alarms improved the model, $F(8, 144) = 11.55, p < .001, \Delta R^2 = .21$. In this revised model, intrusive thoughts and number of false alarms were independent predictors of AH-proneness. Inclusion of the interaction term also improved the model, $F(9, 143) = 11.03, p < .001, \Delta R^2 = .02$. In this revised model, intrusive thoughts, number of false alarms, and the interaction term were independent predictors of AH-proneness.

To interpret the intrusive thoughts \times number of false alarms interaction effect, ModGraph (Jose, 2008)—a programme that graphically displays interaction effects—was employed. As shown in Figure 5.1, when participants reported low levels of intrusive thoughts, they were unlikely to be AH-prone, regardless of the number of false alarms they made. However, as participants reported more intrusive thoughts, strong reality discrimination abilities were associated with a reduction in the risk that participants would be AH-prone. That is, participants who reported high levels of intrusive thoughts, but made few

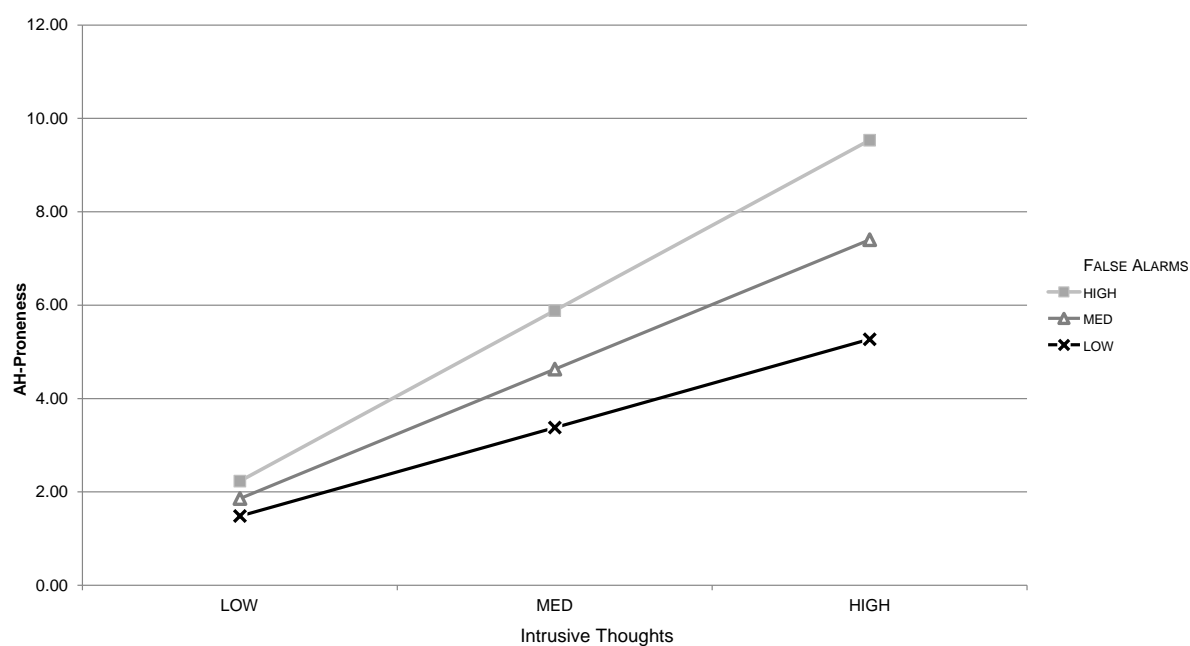
Table 5.2 Summary of hierarchical regression analysis for auditory hallucination-proneness

Variable	B	SE B	β
<i>Block 1</i>			
Age	0.01	0.01	.04
Gender	-0.48	1.43	-.03
Ethnicity	1.47	1.16	.10
Parental income	-2.18	1.52	-.11
Cannabis use	-0.57	1.24	-.04
Negative affect	0.38	0.07	.08***
<i>Block 2</i>			
Age	0.00	0.01	.02
Gender	-1.21	1.25	-.07
Ethnicity	0.30	1.02	.02
Parental income	-1.51	1.36	-.08
Cannabis use	-0.04	1.08	.00
Negative affect	0.14	0.07	.15
Intrusive thoughts	0.57	0.10	.42***
False alarms	0.37	0.10	.25***
<i>Block 3</i>			
Age	0.00	0.01	.02
Gender	-1.13	1.23	-.06
Ethnicity	-0.04	1.02	.00
Parental income	-1.48	1.34	-.07

Cannabis use	-0.05	1.07	.00
Negative affect	0.13	0.07	.15
Intrusive thoughts	0.60	0.10	.42***
False alarms	0.29	0.10	.25***
Intrusive thoughts \times false alarms	0.04	0.02	.15*

* $p < .05$, *** $p < .001$

Figure 5.1 Interaction between level of intrusive thoughts and number of false alarms made during a reality discrimination task.



false alarms, were less likely to be AH-prone than were participants who experienced high levels of intrusive thoughts and made many false alarms.

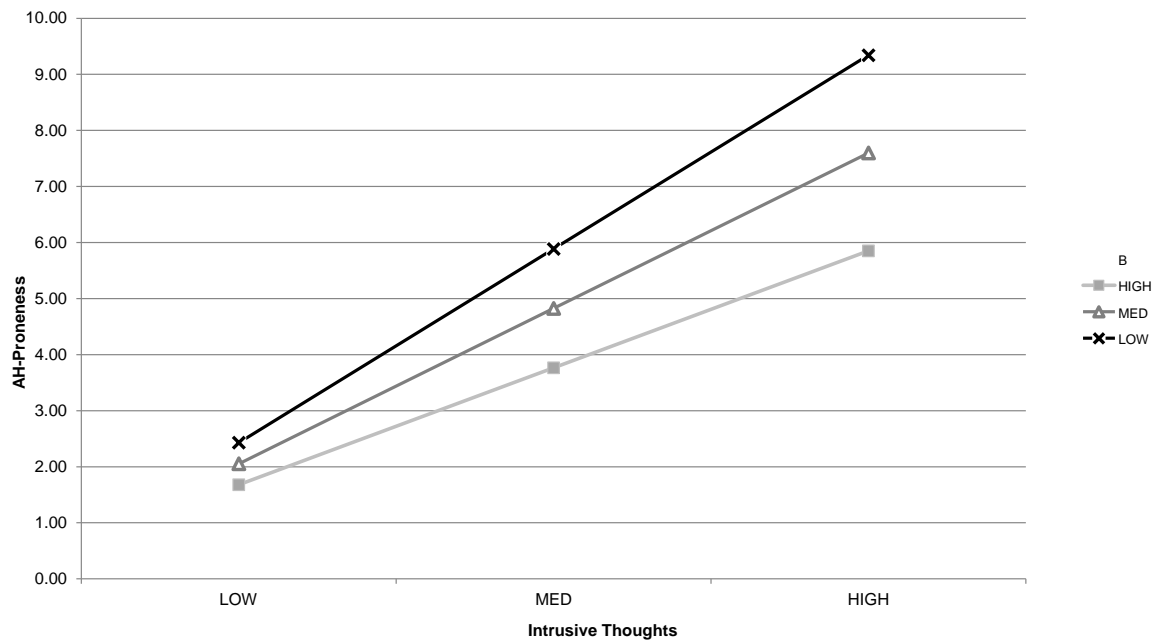
5.4.3 Employing an alternative measure of reality discrimination performance

In the initial analysis, we employed the number of false alarms made as a measure of reality discrimination. However, more commonly, nonparametric β , a representation of participants' response bias, is used. Nonparametric β can vary from 1 to -1. Negative values indicate a more liberal response bias (i.e., a bias towards responding that the speech is present), while positive values indicate a more conservative response bias (i.e., a bias towards responding that the speech is absent). The above regression and moderation analyses were repeated using β as our measure of reality discrimination. β was calculated using the formula presented in Barkus et al. (2007).

The results of the regression analysis were very similar when using β . That is, the inclusion of intrusive thoughts and β as predictor variables in the second step improved the model, $F(8, 144) = 10.23, p < .001, \Delta R^2 = .18$, and both of these variables were independent predictors of AH-proneness. Inclusion of the interaction term in the next step also improved the model, $F(9, 143) = 11.03, p < .001, \Delta R^2 = .01$, but only at a trend level (for $\Delta R^2, p = .09$). In this model, intrusive thoughts and β were independent predictors of AH-proneness, with the interaction term an independent predictor of AH-proneness at a trend level ($p = .09$).

As in the original analysis, ModGraph was used to interpret the interaction effect (see Figure 5.2). When participants reported low levels of intrusive thoughts, they were unlikely to be AH-prone, regardless of their response bias. However, as participants reported more intrusive thoughts, high β values (i.e., a more conservative response bias) were associated with a reduction in the risk that participants would be AH-prone. That is, participants who reported high levels of intrusive thoughts, but had a conservative response bias were less likely to be AH-prone than were participants who experienced high levels of intrusive

Figure 5.2 Interaction between level of intrusive thoughts and β (response bias) during a reality discrimination task.



thoughts and had a liberal response bias. Thus, very similar results were generated when number of false alarms or β were used as a measure of participants' reality discrimination performance.

5.5 Discussion

The present study showed that high levels of intrusive thoughts and weak reality discrimination abilities are independent predictors of AH-proneness. Importantly, the present study showed that these two factors also interact, so that people who report high levels of intrusive thoughts *and* have weak reality discrimination abilities are especially prone to hallucinatory experiences. These findings are consistent with a number of previous studies and with current models of AH, as outlined below.

The association between intrusive thoughts and AH-proneness reported here is consistent with findings from a number of other non-clinical studies, which have also

reported that higher levels of intrusive thoughts are associated with greater hallucination-proneness (Jones & Fernyhough, 2006, 2009). These findings from analogue studies are consistent with data from studies that have employed clinical samples, where psychotic patients with AH reported experiencing more frequent intrusive thoughts in comparison to psychiatric controls (e.g., Lobban et al., 2002; Morrison & Baker, 2000). Together, these studies provide support for the idea that intrusive thoughts may be the raw material of AH, as has been suggested elsewhere (Morrison et al., 1995).

The relation between reality discrimination ability and AH-proneness reported here is concordant with a number of analogue studies (e.g., Barkus et al., 2007; Varese et al., 2011). These studies were part of a recent meta-analysis that reported a moderate-to-large association between reality discrimination performance on the SDT and hallucination-proneness in clinical and non-clinical samples (Brookwell et al., 2013). The zero-order correlation reported here between reality discrimination ability and AH-proneness is broadly similar, although slightly weaker, to the association reported by Brookwell et al., where across nine studies Hedge's $g = 0.87$. Together, these findings support the argument that psychotic patients who experience AH, and those prone to AH, have a predisposition to misattribute internal, self-generated events to an external source, a cognitive bias that is central to most models of AH (e.g., Bentall, 1990; Frith, 1992; Waters et al., 2012).

The present finding of an interaction between reality discrimination errors and intrusive thoughts is consistent with Bentall's (2003) suggestion that a person who experiences high levels of intrusive thoughts *and* who has weak reality discrimination skills will be at especially high risk of developing AH. This finding may also be consistent with Waters, Badcock, Michie, and Mayberry's (2006) 'combined deficit' model, which proposes that some AH are a result of combined deficits in intentional inhibition and context memory. Deficits in intentional inhibition, they argue, result in a person experiencing intrusive

memories over which they feel they have no control. Meanwhile, deficits in context memory mean that when a memory intrudes into consciousness, it lacks various cues that would normally enable a person to identify it as a memory of a past event (e.g., information about the source or the timing of the original event). A person who experiences both of these deficits should therefore experience intrusive, uncontrollable cognitions that do not feel as if they were self-generated memories of past events. These types of cognitions, argue Waters et al. (2006), are likely to be experienced as an AH.

Consistent with their model, Waters et al. (2006) report that in a sample of 43 psychotic patients, 90% of hallucinating patients showed a combination of deficits in intentional inhibition and contextual memory. Only a third of non-hallucinating patients showed this combination of deficits. Given that failures of intentional inhibition lead to the occurrence of intrusive thoughts (e.g., Enright & Beech, 1993), and that Waters et al.'s context memory task involved some aspects of reality discrimination (i.e., participants were asked to recall if they, or an experimenter, had paired two objects together), their combined deficit model may be analogous to the interaction effect reported here. Obviously, however, Waters et al.'s model, and the task they employed, is concerned with a person's ability to determine the source of information from memory, while the task here involved 'on-line' discrimination between self-generated and non-self-generated events. It is thus debatable to what extent Waters et al.'s task and the reality discrimination task employed here measure the same construct (Nelson et al., 2009). Research that examines the associations between the two tasks would help to clarify this issue. In addition, research that examines whether the association between deficits in intentional inhibition and AH-proneness are mediated by intrusive thoughts would be useful. Together, the findings of these studies would indicate to what extent the data reported here are consistent with Waters et al.'s model.

It should be noted that the present findings do not suggest that intrusive thoughts are the only type of mental event that can act as the raw material from which AH are formed. Rather, these results suggest that intrusive thoughts may be one of many types of mental event that can be transformed into a hallucinatory experience. Several authors have argued that inner speech may well form the basis of some AH (e.g., Fernyhough, 2004; Frith, 1992). One possible area for future research is to examine whether the association between AH-proneness and certain types of inner speech is moderated by participants' reality discrimination abilities. For example, recent research has shown that participants who report high levels of dialogic inner speech are more prone to hallucinatory experiences than are participants who report more monologic inner speech (McCarthy-Jones & Fernyhough, 2011). It would be interesting, therefore, to examine whether the associations between dialogic inner speech and AH-proneness are also moderated by participants' reality discrimination abilities.

The present study suffered from a number of limitations. First, the data are cross-sectional, meaning that the causal links between variables are difficult to interpret. There is, as yet, no evidence to show that intrusive thoughts play a causal role in the development of hallucinatory experiences. However, there is some evidence that reality discrimination problems do play a causal role in the development of hallucinatory experiences, as treatments that reduce the frequency of AH also appear to improve psychotic patients' reality discrimination skills (Brunelin et al., 2006; Keefe, Poe, McEvoy, & Vaughan, 2003), supporting the present interpretation of these findings.

Second, the psychometric properties of the hallucination-proneness measure employed here have not yet been established. The most widely-used measure of non-clinical hallucination-proneness is the Launay-Slade Hallucination Scale (LSHS; Launay & Slade, 1981), or various revised versions of the LSHS (e.g., Morrison, Wells, & Nothard, 2000). In

addition to assessing AH-proneness, the LSHS and its revisions assesses a number of different psychological constructs, including visual hallucination-proneness, vividness of daydreams, and frequency of intrusive thoughts (Waters et al., 2012). Recently, a five-item version of the LSHS, which solely assesses predisposition to AH, has been developed (McCarthy-Jones & Fernyhough, 2011). This scale addresses the issue of validity faced by other versions of the LSHS. However, because of its brevity, this scale has relatively low internal reliability ($\alpha = 0.73$; *ibid*). The measure employed here, like McCarthy-Jones and Fernyhough's scale, assesses only AH-proneness, but has higher levels of internal reliability ($\alpha = 0.81$). While further psychometric data on the scale employed here (e.g., establishing test-retest reliability) are required, it may prove to be a more reliable measure of AH-proneness than some existing scales.

Finally, we have employed a non-clinical sample, and so it is unclear whether these findings can be applied to clinical populations. Given that intrusive thoughts are associated with both clinical (Morrison & Baker, 2000) and non-clinical (Jones & Fernyhough, 2009) hallucinatory experiences, and that the same reality discrimination problems are reported in studies that have used clinical and analogue samples (Brookwell et al., 2013), it seems likely that the present findings do apply to the development of clinically-relevant hallucinatory experiences. That said, research examining the associations between intrusive thoughts, reality discrimination, and hallucinatory experiences in a clinical sample is required.

Chapter 6

Study 5: The impact of negative affect on reality discrimination

6.1 Study 5 Pilot

The findings of Study 4 showed that participants' reality discrimination biases are related to hallucination-proneness in a non-clinical sample. Existing research appears to assume that participants' reality discrimination biases are stable and trait-like (e.g., Galdos et al., 2010). However, it is possible that this bias could be modulated, in the same way that other cognitive biases related to the development of psychotic experiences, such as the jumping-to-conclusions bias, can be modulated (e.g., Lincoln, Lange, Burau, Exner, & Moritz, 2010). Thus, the aim of the Study 5 was to examine how participants' reality discrimination abilities might be modulated, using two negative mood inductions. It was predicted that both negative inductions would have an impact on participants' reality discrimination abilities, and this prediction was based on the results of a study not reported in full here (from here on 'Study 5 Pilot'). The rationale for Study 5 Pilot was based on the work of Hoffman (2007) and Pickett and Gardner (2005), who have proposed that periods of social isolation and feelings of loneliness, respectively, create a bias so that people perceive social information to be present when it is not. Moreover, Hoffman has argued that this bias can elicit auditory hallucinations (AH).

One way of testing this idea is by investigating whether feelings of loneliness affect participants' performance on the jumbled speech task (JST). In this task, participants listen to a small amount of reversed, spliced speech, and are asked to report any words or phrases that they perceive in the jumbled speech. When a participant reports hearing a word or a phrase in the jumbled speech, this is referred to as an imaginary verbal experience (IVE; Fernyhough,

Bland, Meins, & Coltheart, 2007) and could be considered an instance of a participant perceiving social information to be present when it is absent. In addition, two studies have reported that the more IVEs participants report, the greater their hallucination-proneness (Campbell & Morrison, 2007; Feelgood & Rantzen, 1994). Therefore, it was predicted that participants who completed a loneliness induction would report more IVEs when performing the JST in comparison to participants who completed a neutral induction, and in comparison to participants who completed another negative induction (which did not involve eliciting feelings of loneliness or social rejection).

Participants were 90 university students (77 women; mean age = 19.72 years, $SD = 0.89$), who were randomly allocated to either a neutral mood induction or one of two negative inductions. In the neutral induction, participants recalled and wrote about their journey to the department that day. In one negative induction, participants recalled and wrote about a time they experienced an academic failure. In the remaining negative induction, participants recalled and wrote about a time they experienced intense feelings of loneliness. After completing an induction, participants began the JST.

At the end of the testing session, participants completed a set of measures that were used to assess whether the inductions had successfully modulated participants' mood. These measures asked participants to report how positive or negative the event recalled was (1 = *very negative*; 7 = *very positive*), the valence of the mood that was generated by recalling the event (1 = *very negative*; 7 = *very positive*), and how the event made them feel about themselves (1 = *very bad about myself*; 7 = *very good about myself*). Consistent with previous research (e.g., Pickett, Gardner, & Knowles, 2004), the failure and loneliness inductions influenced participants' mood. For all three self-report measures of the effect of the mood induction, one-way ANOVA revealed significant group differences: for memory valence, $F(2, 89) = 90.82, p < .001$, for mood after recall, $F(2, 89) = 29.64, p < .001$, and for

how participants felt about themselves after recall, $F(2, 89) = 11.94, p < .001$. Planned contrasts showed that, for all three measures, there were differences between the neutral group and the combined failure and loneliness groups (all $p < .001$). Participants in the failure and loneliness conditions rated the memory they recalled as more negative, rated their mood as more negative after recalling the memory, and reported feeling worse about themselves after recalling the memory than did participants in the neutral condition. Group differences between participants in the failure and loneliness conditions were not significant (all $p > .15$).

Descriptive statistics for JST performance are shown in Table 6.1. Initial analysis indicated that there was no main effect of mood induction on the number of words heard by participants, $F(2, 87) = 0.74, p = .48$. However, following the removal of four outliers, at a trend level, there was a main effect of mood induction on the number of words heard by participants, $F(2, 83) = 2.44, p = .09$. Planned contrasts revealed that, at the trend level, there was a significant group difference between the participants who completed the neutral induction and the participants who completed the two negative inductions, $t(83) = 1.97, p = .05, d = 0.45$. The difference between the participants who completed the failure induction and the participants who completed the loneliness induction was not significant, $t(83) = 0.95, p = .34, d = 0.22$.

Table 6.1 Descriptive statistics for jumbled speech task performance

	Neutral	Failure	Loneliness
Full sample ($N = 90$)			
Mean (SD) number of words heard in the JST	13.43 (9.66)	16.87 (14.69)	15.97 (8.71)
Following the removal of outliers ($N = 86$)			
Mean (SD) number of words heard in the JST	11.25 (5.11)	13.93 (9.81)	15.97 (8.71)

Given that an effect of the mood inductions employed here was found only after the removal of four outliers and, even then, was only significant at a trend level, these findings are far from unequivocal. However, they do suggest that feelings of loneliness, but also negative affect more generally, cause participants' to report more IVEs when performing the JST. In Study 5, therefore, we examined the effect of feelings of loneliness and of negative affect on participants' reality discrimination abilities.

6.2 Abstract

People who experience auditory hallucinations tend to show weak reality discrimination skills, so that they misattribute internal, self-generated events to an external, non-self source. We examined whether inducing negative affect in healthy young adults would increase their tendency to make external misattributions on a reality discrimination task. Participants ($N = 54$) received one of three mood inductions (one positive, two negative) and then performed an auditory signal detection task to assess reality discrimination. Participants who received either of the two negative inductions made more false alarms, but not more hits, than participants who received the neutral induction, indicating that negative affect makes participants more likely to misattribute internal, self-generated events to an external, non-self source. These findings show that negative affect disrupts reality discrimination and suggest one way in which negative affect may lead to hallucinatory experiences.

6.3 Introduction

The process of differentiating between internal, self-generated events and external, non-self-generated events is sometimes referred to as reality monitoring (Bentall, 1990) or reality discrimination (Varese et al., 2011; here we will use the latter term, as the term reality monitoring is more often used in source memory research, e.g., Johnson & Raye, 1981). Cognitive models of auditory hallucinations (AH) suggest that AH occur when internal events (e.g., intrusive thoughts, inner speech) are misattributed to an external agent (e.g., Bentall, 1990; Frith, 1992; Hoffman, 1986). Thus, patients who experience AH should show weak reality discrimination abilities. One way in which reality discrimination abilities are commonly measured in patients with AH is through an auditory signal detection task (SDT; e.g., Barkus et al., 2007). In the SDT, participants must try to detect a signal (typically one second of neutral, non-emotional speech) in an ambiguous auditory stimulus (typically five seconds of white noise). On some trials the speech is present, on other trials the speech is absent. Reality discrimination errors occur when a participant makes a false alarm—that is, when they perceive speech to be present in the white noise when it is absent. Presumably, when a false alarm occurs, participants have mistaken their internal, self-generated representation of the speech for the external, ‘real’ speech. Consistent with current models, when performing a SDT, patients who experience AH show an externalizing bias, whereby they are more likely than controls to report that speech is present in the noise, even when it is absent (e.g., Bentall & Slade, 1985; Varese et al., 2012; Vercammen, de Haan, & Aleman, 2008; Brookwell et al., 2013).

At present, it is unclear why people who experience AH show this externalizing bias. Studies that have examined the antecedents or triggers of AH may suggest some variables that elicit this bias, as presumably problems in reality discrimination peak at times when a person experiences an AH. In Nayani and David’s (1996) study of the phenomenology of

AH, 52% of voice-hearers reported that feelings of sadness preceded the onset of hallucinations, while 16% and 8% reported fear and anger as antecedents, respectively. These findings have been supported by studies that have employed experience sampling methods, which can assess the antecedents and correlates of psychotic experiences in “the flow of daily life” (Myin-Germeys & van Os, 2007, p. 411). In one experience sampling study, participants reported that AH tended to occur in the context of negative affect (Delespaul et al., 2002). Importantly, as these cross-sectional associations might reflect the influence of AH upon mood, Delespaul et al. (2002) also reported that anxiety increased before the onset of AH. This suggests that anxiety (or perhaps negative affect more generally) may play a causal role in the development of AH.

Feelings of loneliness may also modulate reality discrimination. Loneliness is the perception that one’s interpersonal relationships are unsatisfying (Peplau & Perlman, 1982), and it has been shown to be related to, but distinct from, depression and other forms of negative affect (Cacioppo et al., 2006). Psychotic patients have reported that feelings of loneliness (Delespaul et al., 2002) or being alone (Nayani & David, 1996; Tarrier, 1987) precede the onset of AH. Feelings of loneliness tend to elicit high levels of negative affect (Cacioppo et al., 2010) and this may be one way in which loneliness affects reality discrimination. However, it is possible that loneliness also influences reality discrimination through an additional mechanism. Hoffman (2007) has proposed that social isolation (a concept related, but not identical, to loneliness; see de Jong Gierveld, 1998) can lead to a bias where a person begins to attribute social meaning to non-social events and that, in this way, social isolation might play a causal role in the development of AH. For example, high levels of isolation, or intense feelings of loneliness, might encourage an internal, self-generated event, such as inner speech, to be misinterpreted as an external, social event (i.e., as speech directed at you by another person) and this erroneous attribution could form the basis of an

AH. Through this bias, as well as by eliciting negative affect, loneliness may make a person struggle to differentiate internal, self-generated from external, other-generated events.

Therefore, in this study we examined whether experimentally-induced feelings of loneliness, or negative affect more generally, could impair participants' reality discrimination abilities. A mood induction procedure that has been widely used to examine the impact of loneliness on social cognition (e.g., by Pickett et al., 2004) was employed to do this. In this procedure, three inductions are used. All involve participants recalling and writing about an autobiographical memory. One induction requires participants to write about their journey from home to the laboratory and aims to elicit a neutral mood. One induction involves participants recalling a time when they failed at an academic task; this has been shown to elicit negative affect (Pickett et al., 2004). The third induction involves participants recalling a time when they felt intensely lonely; this manipulation has been shown to elicit negative affect and feelings of loneliness (Chen, Williams, Fitness, & Newton, 2008; Pickett et al., 2004). In previous studies, the loneliness induction has influenced a variety of behaviours relating to social cognition (such as gaze orienting and prosody processing), but the failure induction has not influenced these behaviours, despite eliciting negative affect. These findings have been used to support arguments that feelings of loneliness elicit a set of cognitive biases independent of negative affect. Employing this design in the present study allowed us to examine whether there was any effect of negative affect on reality discrimination, and to explore the possibility of an effect of feelings of loneliness on reality discrimination that could be either (a) independent of negative affect, if the failure induction did not influence reality discrimination, or (b) in addition to negative affect, if the failure induction influenced reality discrimination, but to a smaller extent than did the loneliness induction.

In the SDT, several different parameters can be calculated. These include hits (trials where participants correctly report that speech was present in the white noise), false alarms (trials where participants incorrectly report that speech was present in the white noise), sensitivity (which indicates participants' ability to discriminate between trials when speech is present and trials when speech is absent), and response bias (which indicates participants' tendency, across all trials, towards responding that speech is present in the noise). We predicted that participants who received the two negative inductions would make more false alarms, but not more hits, than participants who received the neutral induction. This pattern of results should correspond to lower levels of sensitivity and a more liberal response bias in participants who received the two negative inductions in comparison to participants who received the neutral induction. We also predicted that participants who received the loneliness induction would make more false alarms, demonstrate lower sensitivity, and show a more liberal response bias on the SDT than participants who received the failure induction, as the loneliness induction could elicit an increase in external misattributions via both negative affect and the bias described by Hoffman (2007).

6.4 Method

6.4.1 Participants

Participants were 54 university students (45 women; mean age = 22.08 years, $SD = 5.9$), who received course credit in return for their time. Participants were native English speakers, had normal (or corrected-to-normal) vision, and had no history of hearing problems.

6.4.2 Mood induction

The mood induction described in Study Two of Pickett et al. (2004) was employed here. Participants were randomly assigned to one of three induction groups: a loneliness induction, a failure induction, and a neutral induction. In the loneliness induction, participants

were asked to recall and write down an account of a time when they felt intensely lonely. In the failure induction, participants were asked to recall and write down an account of a time when they experienced an academic failure. In the neutral induction, participants were asked to recall and write down an account of their journey to the department that day. Participants were asked to spend a minimum of five minutes and a maximum of eight minutes on this task. Participants who completed the task in less than five minutes were asked to try to recall more details about their recalled event, and to write about these details. Previous studies have reported that the failure induction effectively elicits negative affect and that the lonely induction effectively elicits both negative affect and feelings of loneliness (Bernstein, Young, Brown, Sacco, & Claypool, 2008; Chen et al., 2008; Maner, DeWall, Baumeister & Schaller, 2007; Pickett et al., 2004; Wilkowski, Robinson, & Friesen, 2009).

6.4.3 Reality discrimination task

A SDT similar to that described by Barkus et al. (2007) was employed to assess reality discrimination. This task consisted of 60 trials, with each trial consisting of five seconds of white noise. In 36 trials, one second of speech was presented in the white noise. In the remaining 24 trials, no speech was presented. In 12 of the trials when speech was presented, the speech was clearly audible. In the remaining 12 trials, the speech was presented at an auditory threshold. This threshold was determined prior to the start of testing by establishing the volume of speech that was perceived by 50% of a small sample ($n = 11$) of participants who were in the same age range as the experimental participants. The stimuli for the speech were prepared from a recording of an adult male reading a piece of non-fictional prose in an emotionally neutral tone. The task was presented to participants on a laptop computer via the experiment software E-Prime 2.0. Participants listened to the task stimuli using standard Sony headphones and responded via a button press at the end of each trial.

The number of hits (trials where participants correctly reported that speech had been present in the white noise) and false alarms (trials where participants incorrectly reported that speech had been present in the white noise) made by participants were recorded. A greater number of false alarms indicated weaker reality discrimination skills. Sensitivity was assessed by calculating d' , which is found by subtracting the z -score of the false alarm rate from the z -score of the hit rate. Higher d' values indicate greater ability to discriminate between trials where speech was present and speech was absent. Response bias was assessed by calculating nonparametric β , as described in Barkus et al. (2007). Nonparametric β can vary from 1 to -1. Negative values indicate a more liberal response bias (i.e., a bias towards responding that the speech is present), while positive values indicate a more conservative response bias (i.e., a bias towards responding that the speech is absent).

6.4.4 Additional measures

Following Pickett et al. (2004), to assess the effectiveness of the mood inductions, participants rated how positive or negative the recalled event was (1 = *very negative*; 7 = *very positive*), the valence of the mood that was generated by recalling the event (1 = *very negative*; 7 = *very positive*), and how the event made them feel about themselves (1 = *very bad about myself*; 7 = *very good about myself*). Participants were also asked to complete the UCLA Loneliness Scale (Russell, 1996) and the Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1983). Inclusion of these measures allowed us to examine whether there were pre-existing group differences in loneliness, depression, or anxiety, and if so, to control for these differences.

6.4.5 Procedure

The study was approved by a departmental ethics committee and was conducted in accordance with the principles of the Declaration of Helsinki. To avoid the demand characteristics associated with some mood inductions (see Buchwald, Strack, & Coyne,

1981), participants were deceived about the true purpose of this study. The study was advertised as research examining links between memory specificity and auditory processing. On arrival at the laboratory, this was reiterated, and participants were told that they would perform the ‘memory specificity task’ first, that this task would involve recalling a memory and writing about it in as much detail as possible, and that an auditory processing task would then be completed. Participants then completed a set of six practice trials for the SDT, to ensure that they understood the task and that they could tolerate the white noise. They were presented with two trials where the speech was clearly audible in the white noise, followed by two trials where the speech was presented at an auditory threshold, and then two trials where the speech was absent. After completing the practice trials, participants confirmed that they could tolerate the white noise and were informed about what type of trials had been presented.

After providing consent, participants were read a set of instructions about the type of memory they were to recall. Participants were randomly allocated to one of the three mood induction. However, participants who were assigned to the failure or loneliness induction, but could not recall a time when they felt intensely lonely, or a time when they felt they had failed at an academic task, were re-assigned to the neutral induction. Four participants were re-assigned from the failure induction and five participants were re-assigned from the loneliness induction. Following completion of the SDT, participants were presented with the self-report measures. Following completion of these measures, participants were informed that they had been deceived about the true purpose of the study, were asked whether they suspected that they had been deceived, and were fully debriefed. None of the participants guessed the true nature of the study. They were then invited to ‘repair’ their mood by watching a short clip of their choice from a variety of comedy television series.

6.4.6 Statistical analysis

Gender differences were assessed using *t*-tests. A series of one-way ANOVAs were used to investigate the effect of the mood inductions. Where appropriate, ANOVA was followed by planned contrasts. In all instances, the first contrast was between mean scores in the neutral group versus the combined means of the two negative induction groups, with the second contrast between the means of the failure and loneliness groups.

6.5 Results

6.5.1 *Loneliness, depression, and anxiety*

Mean scores for loneliness, depression, and anxiety are presented in Table 6.2. Group differences in levels of depression, anxiety, and loneliness were not significant (all *F*-values < 1.5 , all $p > .24$) and so these variables are not considered in any of the subsequent analyses.

6.5.2 *Gender differences*

Given the unbalanced nature of the sample, we investigated the influence of gender on number of false alarms, number of hits, d' , and β values. When looking at the whole sample, gender differences were not significant (all $p > .14$), except for a trend level difference in d' , $t(52) = 1.74$, $p = .09$, $d = 0.62$. Men ($M = 1.80$, $SD = 0.54$) had marginally higher d' values than did women ($M = 1.45$, $SD = 0.56$). However, this difference appears to be a function of the large proportion of men (five of nine) who were assigned to the neutral group. When gender differences were examined within each mood induction group, there were no differences between men and women (all $p > .28$). Gender is thus not considered in the subsequent analyses.

6.5.3 *Manipulation check*

Mean scores for the self-report manipulation check scales are presented in Table 6.2. For two of the three self-report measures of the effect of the mood induction, one-way ANOVA revealed significant group differences: for memory valence, $F(2, 51) = 12.43$, $p < .001$, and for mood after recall, $F(2, 51) = 15.26$, $p < .001$. In addition, at trend level, there

Table 6.2 Descriptive statistics for all study variables

	Neutral	Failure	Loneliness
<i>Reality discrimination performance</i>			
Number of false alarms	3.22 (3.51)	7.17 (4.64)	5.33 (4.50)
Number of hits	24.44 (3.17)	25.67 (3.01)	25.11 (4.75)
d'	1.79 (0.56)	1.23 (0.55)	1.50 (0.49)
β	0.46 (0.44)	0.12 (0.37)	0.23 (0.48)
<i>Ancillary measures</i>			
Loneliness	39.50 (7.57)	37.28 (8.13)	38.50 (7.36)
Depression	5.17 (4.05)	4.17 (2.83)	3.33 (2.66)
Anxiety	9.33 (4.64)	8.61 (3.45)	8.33 (4.43)
<i>Manipulation checks</i>			
Mean memory valence (SD)	4.70 (1.05)	2.17 (0.87)	1.83 (0.75)
Mean mood after recall (SD)	5.03 (1.13)	3.43 (1.01)	3.23 (0.82)
Mean felt about self after recall (SD)	4.53 (0.82)	3.43 (0.90)	3.73 (0.98)

was a main effect of induction on how participants felt about themselves after recall, $F(2, 51)$, $= 3.12$, $p = .06$. Planned contrasts showed that, for all three measures, there were differences between the neutral group and the loneliness and failure groups. Participants in the loneliness and failure conditions rated the memory they recalled as more negative, rated their mood as more negative following the recall task, and reported feeling worse about themselves following the recall task than did participants in the neutral condition (all $p < .05$). Group differences between participants in the loneliness and failure conditions were not significant (all $p > .41$).

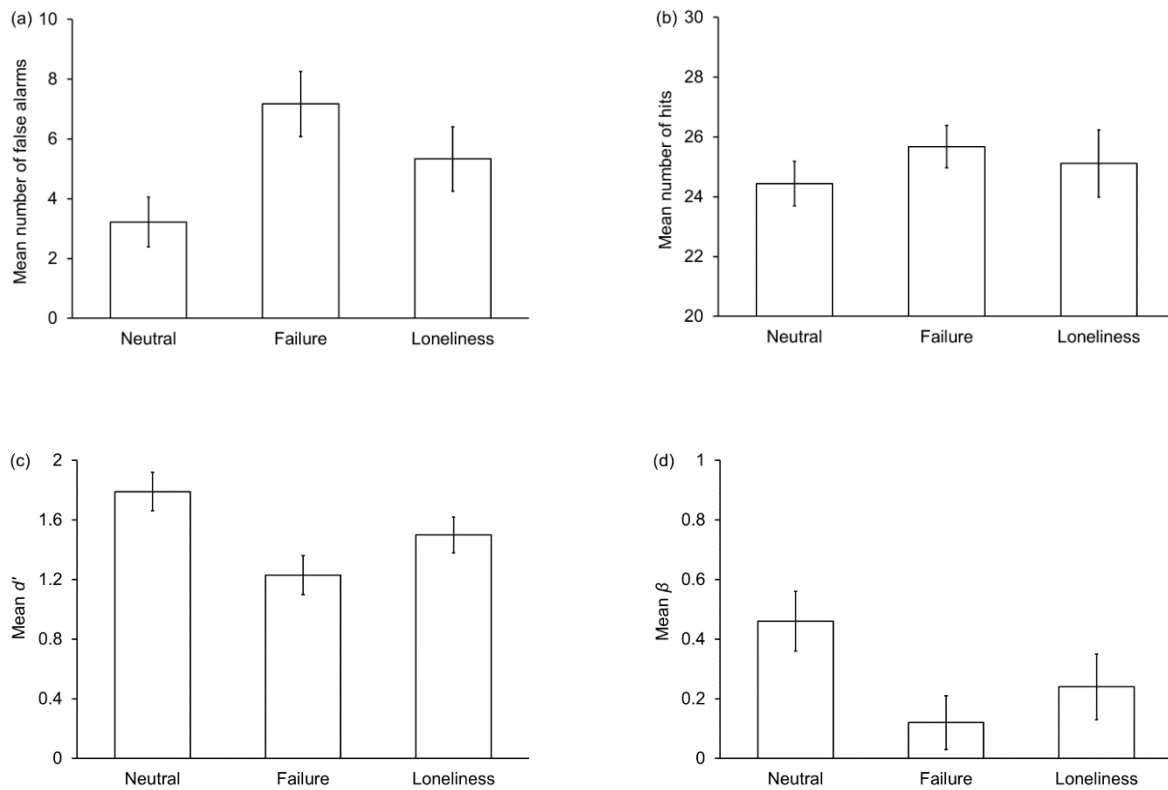
6.5.4 SDT performance

Descriptive statistics for the measures of SDT performance are shown in Table 6.2 and are also presented in Figure 6.1. There was a main effect of induction on the number of false alarms participants made, $F(2, 51) = 3.83, p = .028$. Participants in the failure group made the most false alarms, followed by participants in the loneliness group, with participants in the neutral group making the fewest false alarms. The first planned contrast revealed that the neutral group made fewer false alarms than participants in the two negative inductions, $t(51) = 2.45, p = .018, d = 0.75$. The second planned contrast revealed that the difference in number of false alarms between the failure and loneliness groups was not significant, $t(51) = 1.29, p = .20, d = 0.39$. In addition, this non-significant group difference was in the direction opposite to that predicted. In contrast, with respect to number of hits, there was no effect of induction, $F(2, 51) = 0.49, p = .62$.

There was a main effect of induction on d' , $F(2, 51) = 4.88, p = .012$. d' values were lowest in the failure group, followed by the loneliness group, with d' values highest in the neutral group. The first planned contrast revealed that d' was higher in the neutral group than in the two negative induction groups, $t(51) = 2.72, p = .009, d = 0.73$. The second planned contrast revealed that the difference in d' between the failure and loneliness groups was not significant $t(51) = 1.54, p = .13, d = 0.51$. Again, this non-significant group difference was in the direction opposite to that predicted (d' values were predicted to be lower in the loneliness group).

Finally, at the trend level, there was a main effect of induction on β , $F(2, 51) = 3.02, p = .06$. β values were lowest in the failure group, followed by the loneliness group, with β values highest in the neutral group. The first planned contrast revealed that β was higher in the neutral group than in the two negative induction groups, $t(51) = 2.32, p = .024, d = 0.65$. The second planned contrast revealed that the difference between the failure and loneliness groups was not significant $t(51) = 0.80, p = .42, d = 0.27$. Again, this non-significant group

Figure 6.1 Mean (a) number of false alarms, (b), number of hits, (c), d' and (d) β in the three mood induction groups.



difference was in the direction opposite to that predicted (β values were predicted to be lower in the loneliness group).

6.6 Discussion

The present study set out to examine whether experimentally-induced negative affect and feelings of loneliness could elicit an externalizing bias when participants performed a reality discrimination task. Participants who received either of the negative inductions made more external misattributions than did participants who received a neutral mood induction. Importantly, the negative inductions did not appear to impair participants' performance in all aspects of the task. That is, participants who received the two negative inductions made the same number of hits as did participants who received the neutral induction. This indicates that the negative inductions did not impair participants' ability to detect a signal when it was

present, but that they specifically made participants more likely to misattribute internal, self-generated events to an external, non-self source.

If one assumes that a person's reality discrimination skills are weakest when they experience AH, these findings can be considered consistent with a number of studies. Nayani and David (1996), Tarrier (1987), and Delespaul et al. (2002) have all reported that some form of negative affect tends to occur around the onset of AH. While Nayani and David's and Tarrier's studies relied on retrospective reporting, Delespaul et al.'s experience sampling data provided evidence that anxiety precedes the onset of AH, suggesting that negative affect plays a causal role in the day-to-day onset of AH in psychotic patients. The present findings are consistent with this suggestion and they indicate one mechanism by which negative affect can cause AH. It is possible that negative affect might lead to AH through other mechanisms (e.g., by increasing the likelihood that a person will experience intrusive, unpleasant thoughts that are difficult to identify as internal, self-generated events) and future research should examine whether this is the case.

More broadly, by demonstrating that negative affect elicits a bias considered to be important in the development of AH, these findings are consistent with approaches that have emphasized the importance of affective problems in the development of psychotic experiences (Freeman & Garety, 2003). These approaches (e.g., Garety et al., 2001) typically focus on the way in which emotion may influence the content of AH (e.g., depression may cause a person to hear a voice telling them that they are worthless) or on the way in which a person responds to a hallucinatory experience (e.g., anxiety may cause a person to respond to a threatening voice in a fearful, subordinate manner). The present study, however, suggests that these accounts should also consider the possibility that emotional problems may elicit the biases that help to trigger AH.

The present findings could be considered to be inconsistent with Hoffman's (2007) suggestions for the role social isolation might play in the development of AH. Hoffman has argued that social isolation creates a bias so that a person will begin to attribute social meaning to non-social events. Given the associations between loneliness and social isolation (e.g., Golden et al., 2009), it seems likely that loneliness will encourage a person to attribute social meaning to non-social events. And, given that internal, self-generated events tend to be non-social while external, non-self-generated events may often be social (e.g., they may be instances of another person talking to you), we predicted that loneliness would elicit an externalizing bias. While participants who completed a loneliness induction did show an externalizing bias in comparison to control participants, they did not show a greater externalizing bias than participants who completed a negative affect induction. This suggests that while loneliness can elicit an externalizing bias, it does so via negative affect, rather than through the mechanism suggested by Hoffman. It could be argued that this finding is simply a result of employing an ineffective loneliness induction. However, this induction has been used successfully in a range of studies to elicit feelings of loneliness and a set of biases associated with high levels of loneliness (e.g., Chen et al., 2008; Wilkowski et al., 2009), and so this seems unlikely. Rather, given that Hoffman's hypothesis focuses on social isolation, not loneliness, it is possible that only a procedure that involves isolating participants from human contact, rather than simply asking them to recall a time when they felt isolated from others, would elicit the kind of bias Hoffman described. Future research should thus examine the effects of social isolation on reality discrimination and other aspects of self-monitoring.

Negative affect may have elicited an externalizing bias in this study through a number of different mechanisms. One possibility is that strong reality discrimination abilities rely upon intact working memory, that negative affect interferes with working memory capacity, and in this way impairs reality discrimination. This interpretation is suggested by research

showing that (a) participants are less likely to identify themselves as the agent of an action (and so will presumably be more likely to display an externalizing bias) when working memory load is increased (Hon, Poh, & Soon, 2013) and (b) negative affect reduces working memory capacity (Elzinga & Roelofs, 2005; Schoofs, Preuss, & Wolf, 2008). Research that examines whether impairments in working memory mediate the effect of negative affect on reality discrimination is required.

In sum, the present study showed that negative affect can elicit biased reality discrimination in a non-clinical sample of young adults. Increases in negative affect typically occur prior to the onset of AH in the daily lives of psychotic patients, and the present findings suggest that this association may be explained in terms of the influence of negative affect on reality discrimination. This finding requires replication in a clinical sample, but is consistent with approaches that emphasize the importance of emotion in the development of psychotic experiences.

Chapter 7

General Discussion

7.1 Summary of findings

The studies in this thesis investigated the roles played by parental bonding, attachment anxiety and avoidance in peer relationships, and reality discrimination in the development of two types of psychotic-like experience (PLE)—non-clinical auditory hallucinations (AH) and non-clinical paranoid thinking. Given that there is evidence that the study of PLEs is valuable in understanding the aetiology of AH and paranoid thinking experienced by psychotic individuals (Kaymaz et al., 2012; Kelleher & Cannon, 2011), the findings outlined below should have implications not only for understanding the development of PLEs, but also for understanding the development of clinically-relevant psychotic experiences.

7.1.1 Parental bonding and PLEs

In Study 1, associations between parental bonding, negative affect, bullying, and PLEs were examined in a sample of adolescents. Previous research has reported reliable associations between poor parental bonding and psychosis (e.g., Parker et al., 1982) and there is some evidence that these relations may be mediated by subsequent adversity (e.g., emotional, physical, or sexual abuse; Janssen et al., 2005) and/or by negative affect (e.g., Bebbington et al., 2004). In Study 1, only one aspect of parental bonding—maternal care—was found to be an independent predictor of paranoid thinking and of AH-proneness. Consistent with previous research, it was found that associations between maternal care and both types of PLE were mediated by negative affect and by bullying.

7.1.2 Attachment style and PLEs

In Study 2, associations between attachment anxiety, attachment avoidance, loneliness, and PLEs were examined in a non-clinical sample of adults. It has been proposed that social isolation, a construct closely related to loneliness, plays a direct causal role in the development of psychotic experiences (Hoffman, 2007). Given the associations between peer attachment style and loneliness (e.g., DiTommaso et al., 2003; Wei et al., 2005) and between social isolation and loneliness (de Jong Gierveld, 1998), it was argued that loneliness may mediate the relations between PLEs and both attachment anxiety and attachment avoidance. It was found that loneliness mediated the associations between attachment anxiety and paranoid thinking and between attachment avoidance and paranoid thinking. AH-proneness was not associated with attachment anxiety, attachment avoidance, or loneliness.

In Study 3, associations between attachment anxiety, attachment avoidance, anomalous percepts, and paranoid thinking were examined in a non-clinical sample of adults. A popular model of paranoid thinking (Freeman, 2007) posits that persecutory beliefs are a rational attempt to explain anomalous experiences (e.g., “My food tastes strange. Someone must be trying to poison me”), but this model does not provide an explanation of why a person would misinterpret an anomalous percept as an indicator of social threat. Individuals who experience high levels of attachment anxiety are hypothesized to perceive innocuous events as indicators of social threat (Mikulincer et al., 2003). It was argued, therefore, that attachment anxiety would moderate the association between anomalous percepts and paranoid thinking. Consistent with this prediction a moderating effect was found, such that when participants experienced infrequent anomalous percepts, those who reported high levels of attachment anxiety were more likely to report paranoid thinking than were those who reported low levels of attachment anxiety. Individual differences in attachment avoidance did not moderate this association, indicating that there was not a broad effect of insecure attachment, only a specific moderating effect of attachment anxiety.

In sum, across these three studies, some of the mechanisms through which poor parental bonding and attachment anxiety and/or avoidance may relate to PLEs have been demonstrated. Given the aetiological links between PLEs and clinically-relevant psychotic experiences (Kelleher & Cannon, 2011), it is possible that these are the same mechanisms involved in AH and paranoid thinking in psychotic individuals.

7.1.3 Reality discrimination and AH

In Study 4, associations between intrusive thoughts, biased reality discrimination abilities, and AH-proneness were examined in a non-clinical sample of adults. Previous research has reported associations between intrusive thoughts and AH-proneness (Jones & Fernyhough, 2006, 2009) and between biased reality discrimination abilities and AH-proneness (Brookwell et al, 2013), but no study has yet looked at interactions between intrusive thoughts and reality discrimination abilities. Following Bentall (2003), it was predicted that the association between intrusive thoughts and AH-proneness would be moderated by participants' reality discrimination abilities, and this proved to be the case. Participants who made frequent external misattributions when performing a reality discrimination task and who reported high levels of intrusive thoughts were especially likely to be prone to AH.

In Study 5, the effects of induced negative affect and of induced feelings of loneliness on participants' performance on a reality discrimination task were examined. Negative affect was induced by asking participants to recall and write about a time they had failed at an academic task, or about a time they had experienced intense feelings of loneliness. It was predicted that participants who received either induction would make more frequent external misattributions than participants who received a control induction, and this proved to be the case. This finding is in line with the reports of patients who experience AH, as they report that some form of negative affect (e.g., anxiety, anger, fear, and sadness) often precedes the

onset of AH (Delespaul et al., 2002; Nayani & David, 1996; Tarrier, 1987), suggesting that negative affect may elicit AH by impairing a person's ability to distinguish between internal, self-generated and external, non-self-generated events.

7.1.4 Perceived parental income and PLEs

Three of the studies reported here included an assessment of perceived parental income during childhood, as a proxy for childhood socio-economic status (SES). This measure was employed as it is brief, which was important for practical reasons, and because it has been reported that this measure accounts for more variance in symptoms of psychopathology than objective measures of childhood SES, such as actual parental income (Teicher et al., 2010). In the three studies where it was assessed, participants were divided into one group who reported that their parents' income was less than, or much less than enough to meet their needs, and a second group who reported that their parents' income was enough, more than enough, or much more than enough to meet their needs. In each study, preliminary analysis compared levels of PLEs in these two groups and in subsequent regression analyses this variable was entered as a covariate. In Study Two, participants who perceived that their parents' income met their needs reported lower levels of paranoid thinking than did participants who reported that their parents' income was not enough to meet their needs. However, in a regression analysis, perceived parental income was not a significant predictor of paranoid thinking. In addition, in this study, perceived parental income was unrelated to AH-proneness. In Study Three, at a trend level (i.e., $p = .06$), participants who perceived that their parents' income met their needs reported lower levels of paranoid thinking than did participants who reported that their parents' income was not enough to meet their needs. In a regression analysis, perceived parental income remained a significant predictor, accounting for around 3% of the variance in paranoid thinking. In Study Four, perceived parental income was unrelated to levels of AH-proneness.

Thus, across these three studies, there appears to be no association between perceived parental income and AH-proneness. However, there does appear to be an association between perceived parental income and paranoid thinking, although this may not persist after controlling for other variables. These findings are broadly consistent with studies that have reported associations between socio-economic disadvantage and psychotic experiences (e.g., Agerbo et al., 2004; Harrison et al., 2001). In addition, these results are consistent with the claim that different types of social adversity are associated with different psychotic experiences (Bentall & Fernyhough, 2008). More specifically, they could be considered to be consistent with the idea that while paranoid thinking is associated with exposure to experiences of powerlessness and discrimination (given the strong associations between poverty and powerlessness; Chambers 1995), AH are associated with exposure to much more intrusive and traumatic experiences, such as childhood sexual abuse (Bentall et al., 2012). While, socio-economic factors, like parental income, clearly play an important role in the development of psychotic experiences, the remainder of this Discussion will focus on the roles of parental bonding, attachment style, reality discrimination, and negative affect in the development of AH and paranoid thinking.

7.2 Theoretical implications

The five studies reported here are broadly consistent with current psychological models of psychotic experiences. For example, a number of models have proposed that problematic parental or peer attachment relationships play a role in the development of psychosis (e.g., Bentall et al., 2007). These models (Bentall et al., 2001; Berry et al., 2007) tend to propose that attachment factors increase the risk for psychosis by influencing a person's schematic beliefs about themselves (i.e., poor parental bonding leads to negative beliefs about the self) or about others (i.e., beliefs about the untrustworthiness of others, in part, underlie attachment anxiety). The findings presented in Studies 1, 2, and 3, however,

suggest that a number of other mechanisms—negative affect, exposure to bullying, feelings of loneliness, and a tendency to misinterpret anomalous percepts as indicators of social threat—should also be considered.

Meanwhile, a number of cognitive models propose that reality discrimination biases may underlie AH (Beck & Rector, 2003; Bentall, 1990; Waters et al., 2012). The findings presented here are consistent with these models, in that associations between biased reality discrimination abilities and AH-proneness were reported. The present findings also provide the first empirical evidence that biased reality discrimination skills moderate the association between intrusive thoughts and AH-proneness, which has been suggested by Bentall and colleagues (2003; Bentall et al., 2007). This type of interaction effect emphasizes the value of multi-factorial models of AH, in that both the presence of biased reality discrimination skills and of intrusive thoughts appear to be important in explaining AH-proneness, at least in a non-clinical sample.

The findings presented in Study 5—that reality discrimination biases can be elicited through the induction of negative affect—suggest ways in which models of AH could be extended. Some previous studies appear to have assumed that the cognitive biases associated with psychotic experiences are stable (e.g., Buchy, Woodward, & Liotti, 2007; Galdos et al., 2010). However, the modulation of reality discrimination reported here suggests that these biases can fluctuate. This is consistent with evidence showing that AH fluctuate in patients' daily lives. For example, interview-based studies with patients who experience AH have shown that the majority of patients do not report AH constantly (Nayani & David, 1996; McCarthy-Jones et al., 2012). Similar findings have been reported by studies that have employed experience sampling methods to assess the occurrence of AH in the “flow of daily life” (Myin-Germeys & van Os, 2007, p. 411). Across a number of experience sampling studies, patients report experiencing AH on 30-40% of the occasions that they are asked to

respond (Delespaul et al., 2002; Oorschot et al., 2012; Peters et al., 2012). It is clear, therefore, that in patients' daily lives there is a great deal of fluctuation in AH.

At present, however, cognitive models of AH do not provide detailed accounts of why one would expect these fluctuations. For example, Gallagher (2004) has argued that cognitive models of AH fail to explain why not all thought is experienced as an AH. In showing that reality discrimination biases can be modulated, the results of Study 5 can account for why there is daily fluctuation in AH, and this finding could be incorporated into existing cognitive models of AH. This would enable these models to provide a partial account of why AH fluctuate, and this could be part of a larger focus on explaining why these fluctuations occur. This would be valuable for a number of reasons. Better accounts of why AH fluctuate would be helpful in that they would address the concerns of Gallagher. In addition, better accounts of why AH fluctuate might also bring about more practical benefits. For example, more focus on the factors that trigger the onset of AH may encourage the development of a wider set of evidence-based strategies that reduce the frequency of AH. This may prove to be a valuable adjunct to cognitive-behavioural therapies for psychosis, which focus on reducing the distress associated with the occurrence of AH (Morrison, 2004), rather than on ways to reduce the frequency of AH.

The findings of the five studies reported in this thesis are consistent with approaches that view psychotic experiences as endpoints of atypical social, cognitive, and emotional developmental trajectories. Importantly, the studies reported in this thesis have shown that social (e.g., parental bonding, attachment style, bullying, and loneliness), cognitive (e.g., anomalous perceptual experiences, reality discrimination biases, intrusive thoughts), and emotional (e.g., negative affect) factors do not act independently of one another. In some instances, these factors may be mediated by one another. For example, the findings of Study 1 suggested that low levels of maternal care can elicit high levels of negative affect and, in

doing so, place a person at risk of developing psychotic experiences. Meanwhile, the findings of Study 5 showed that high levels of negative affect can lead to biased reality discrimination, which is thought to predispose a person towards experiencing AH. Alternatively, in other instances, these factors may be moderated by each other. For example, the findings of Study 3 showed that individual differences in attachment anxiety moderate the association between anomalous percepts and paranoid thinking. Meanwhile, the findings of Study 4 showed that reality discrimination biases moderate the association between experiencing intrusive thoughts and AH-proneness. The ways in which these factors interact with one another in the development of AH and paranoid thinking are depicted in Figure 7.1 and Figure 7.2, respectively.

7.3 Directions for future research

A number of recommendations for future research are immediately apparent. First, it will be important to replicate these studies in clinical samples. This is because, while all of the variables examined in the studies described in this thesis have been shown to be associated with both clinical and non-clinical psychotic experiences, it is possible that the moderating or mediating effects reported here may not be found when studies examine AH and paranoid thinking in clinical samples. For example, a number of studies that have employed clinical and non-clinical samples have reported associations between low levels of maternal care and psychotic experiences (Parker et al., 1982; Meins et al., 2008) and between bullying and psychotic experiences (Schreier et al., 2009; Trotta et al., 2013). However, this does not necessarily mean that the finding reported in Study 1—that exposure to bullying partially mediates the association between low maternal care and psychotic experiences in non-clinical participants—will be replicated in a clinical sample. This is possible for a number of reasons. First, it is possible that clinical and non-clinical psychotic experiences are

Figure 7.1 A model of AH consistent with the data presented in this thesis

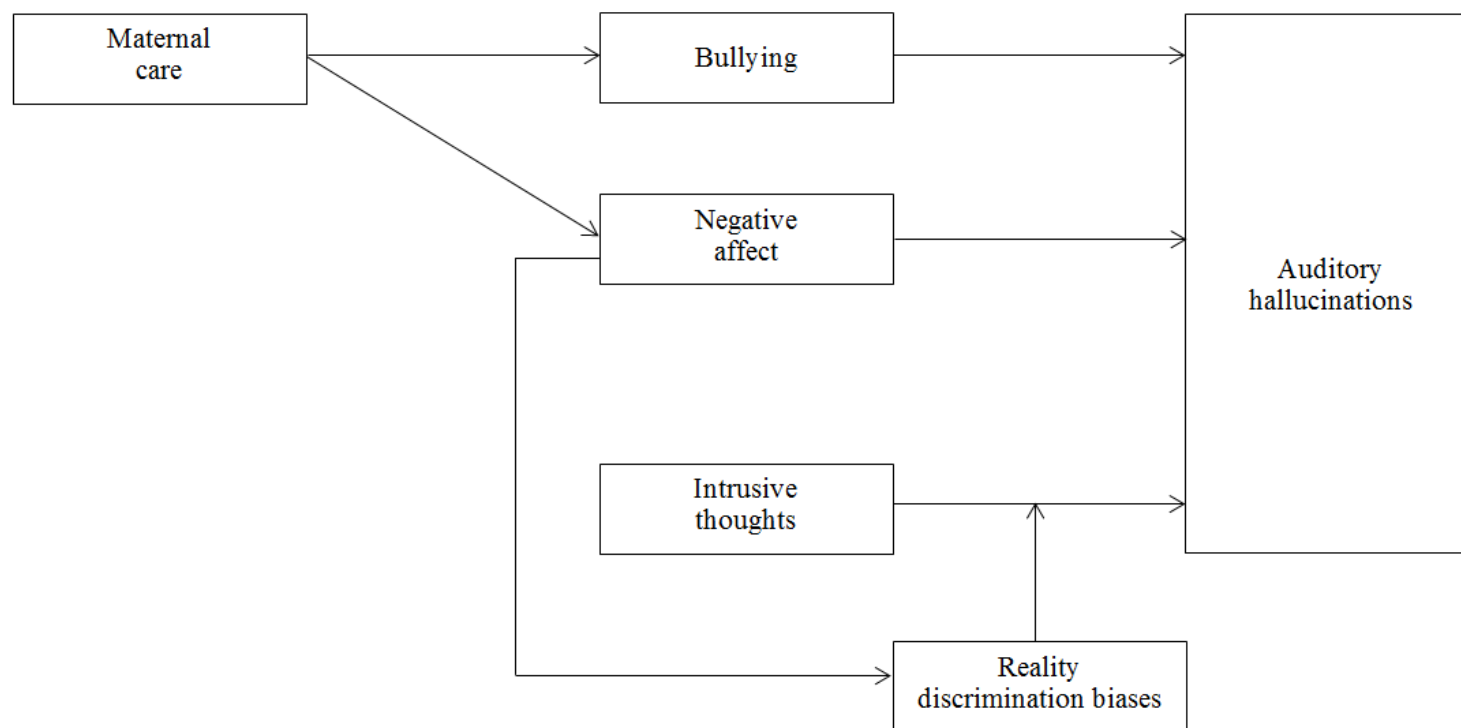
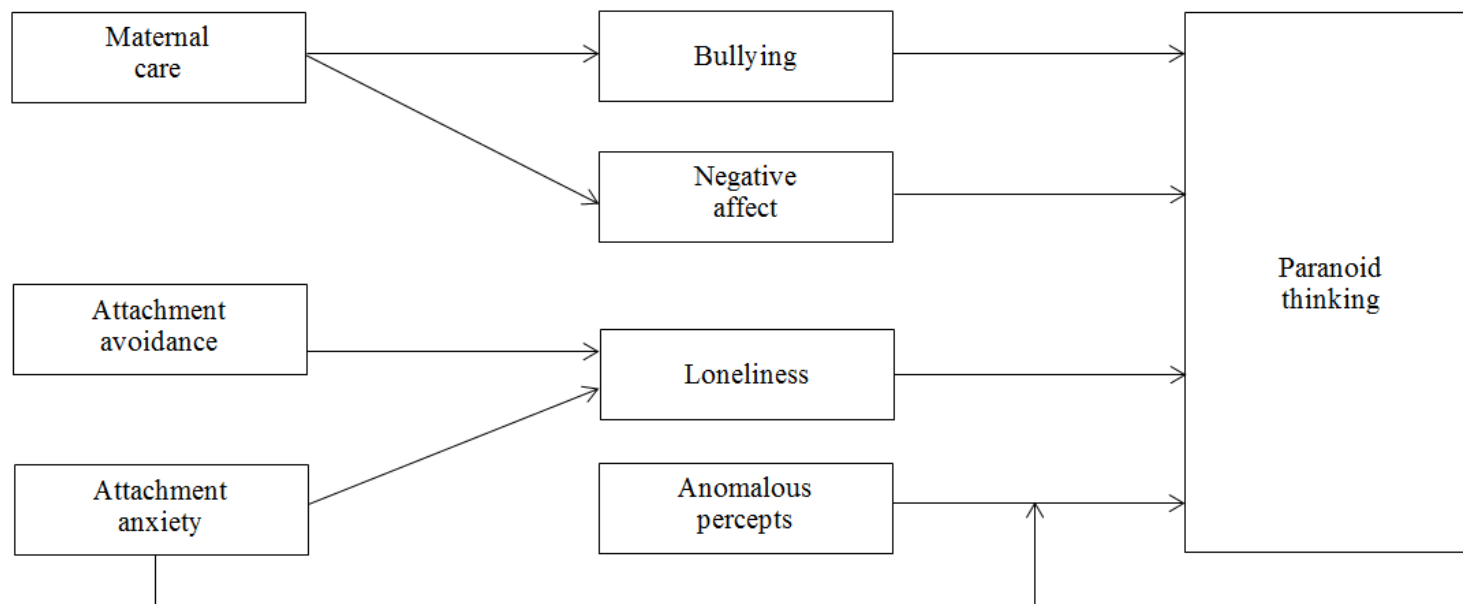


Figure 7.2 A model of paranoid thinking consistent with the data presented in this thesis



the same phenomena (Esterberg & Compton, 2009), but that the need for care associated with clinical psychotic experiences means that there will be at least some differences in the development of these experiences. Alternatively, Badcock and Hugdahl (2012) have argued that clinical and non-clinical psychotic experiences may be fundamentally different phenomena. For example, there are important affective and phenomenological differences between clinical and non-clinical psychotic experiences, with clinical psychotic experiences identified as being more negative in content and as more often taking the form of a male voice. These differences may, according to Badcock and Hugdahl, point towards different mechanisms underlying clinical and non-clinical psychotic experiences. Meanwhile, Daalman et al. (2011) have shown that participants with clinically-relevant AH report that they began to experience AH at a later age than do participants with AH that are not clinically relevant. This suggests that the development trajectories of clinical and non-clinical psychotic experiences may differ, and this may mean that they are associated with different causal mechanisms. It remains unclear, therefore, to what extent clinical and non-clinical psychotic experiences share the same causal mechanisms, and so it will be important to replicate the findings presented in this thesis in clinical samples.

Second, longitudinal studies that are better able to establish the causal relations between the variables studied here are also required. Four of the five studies reported here employed cross-sectional designs, which can tell us little about the causal links between the variables under study. Longitudinal or experimental research has supported a causal role for attachment style (e.g., Berry et al., 2008), bullying (e.g., Schreier et al., 2009), negative affect (e.g., Krabbendam et al., 2002), loneliness (e.g., van der Werf et al., 2010), and anomalous percepts (Zimbardo et al., 1981) in the development of psychotic, or psychotic-like, experiences. However, little research has examined how these variables interact with one another over time to lead to the development of psychotic experiences, and such research is

required if the developmental trajectory by which psychotic experiences emerge is to be properly described. Such research would require a large adolescent sample (as this is the age when exposure to bullying peaks; Nansel et al., 2001), who would complete multiple assessments over a number of years. If the present interpretation of how AH and paranoid thinking develop is correct, one would expect changes in attachment style, in exposure to bullying, and in the presence of anomalous percepts to prospectively predict changes in PLEs in adolescents. In addition, one would expect the associations being attachment anxiety and avoidance to be mediated by changes in loneliness, and that the association between anomalous percepts and paranoid thinking would be moderated by attachment anxiety.

More broadly, as discussed in the General Introduction, four of the five studies reported in this thesis examined the predictors of PLEs. The argument that such an approach is valuable in understanding the development of clinically relevant AH and paranoid thinking rests on the assumption that there is a continuum of psychotic experiences throughout the general population. There is good evidence to support this assumption (e.g., Kaymaz et al., 2012; Linscott & van Os, 2013), although, as discussed above, research that replicates the associations between PLEs in clinical sample is required. In addition to that research, recent work on the nature of the continuum of psychotic experiences (e.g., Kaymaz & van Os, 2010) suggests other avenues for future research. These revisions focus on the claim that there may be multiple continua, rather than a single continuum, of psychotic experiences.

As proposed by Johns et al. (submitted), two continua exist, with one continuum composed of people who are genetically predisposed to psychosis and a second continuum of people who are not. In those who are genetically predisposed to psychosis, AH and paranoid thinking are proposed to be related to cognitive impairment and structural brain atypicalities, which may have been present at birth. Psychotic experiences in this group tend to be associated with a greater likelihood of need for care and poorer outcome, as the

neurobiological problems that are thought to underlie these experiences appear to be very difficult to alleviate (Myin-Germeys & van Os, 2007). In contrast, in those who are not genetically predisposed to psychosis (the ‘affective’ sub-type), AH and paranoid thinking are proposed to be related to affective problems and neurochemical atypicalities. Psychotic experiences in this group tend to be associated with a lower likelihood of need for care and with better outcomes, as the affective problems and functional brain changes (e.g., hyperdopaminergic activity) that are thought to underlie these experiences are more amenable to currently available treatments (Myin-Germeys & van Os, 2007). If the existence of these sub-types is confirmed, it will be important to examine whether the assumption that there is a continuum of clinical and non-clinical psychotic experiences is valid in both sub-types. That is, it may prove to be the case that, while in one group the same mechanisms cause both clinical and non-clinical psychotic experiences, in the second group there may be discontinuity in the mechanisms that cause clinical and non-clinical experiences. Research that examines whether this is the case is required.

Moreover, it will be of interest to examine the developmental trajectories of AH and paranoid thinking in these two sub-types. It is clear that these trajectories will differ to some extent (i.e., one group is thought to have a genetic predisposition to psychosis, the other group is not), but there may also be points of considerable convergence. For example, one might find that in both the genetically-prone sub-type and in the affective sub-type, AH-proneness is a result of a combination of biased reality discrimination and a tendency to experience intrusive thoughts. However, the two groups might differ in the ways in which these intrusive thoughts have developed. In the genetically-prone sub-type, intrusive thoughts may be a result of genetic factors (as in some cases of obsessive-compulsive disorder; Clifford, Murray, & Fulker, 1984), while in the affective sub-type, intrusive thoughts may be a result of social adversity (as in post-traumatic stress disorder; Ehlers & Clark, 2000).

Research that examines to what extent there is overlap in the mechanisms underlying the development of psychotic experiences in these hypothesized sub-types will be valuable.

In terms of parental bonding, further research examining the ways in which low levels of maternal care may lead to the development of psychotic experiences would be useful. A number of studies have shown that participants who report receiving low levels of maternal care during childhood tend to have smaller hippocampal volumes than participants who report high levels of maternal care (Engert et al., 2010; Narita et al., 2012). Smaller hippocampal volumes are observed in patients with schizophrenia (Wright et al., 2000), and abnormal activity in the hippocampal formation is associated with the onset of AH (Jardri & Sommer, 2013). It has been argued that this abnormal hippocampal activity results in perceptual information that is drawn from memory failing to be identified as self-generated, and so is experienced as a hallucination (Diederer et al., 2010). These findings might suggest that low levels of maternal care lead to atypical development of the hippocampus, and that this increases the risk that a person will develop AH. Future research could, thus, investigate whether hippocampal abnormalities mediate the association between maternal care and AH-proneness.

The association between exposure to frequent bullying and PLEs reported in Study 1 is consistent with a growing body of evidence that bullying plays a causal role in the development of psychotic experiences (e.g., Arseneault et al., 2011; Schreier et al., 2009). Recent research has identified an external locus of control and low self-esteem as two factors that mediate the association between bullying and PLEs in adolescents (Fisher et al., 2013). Another possibility is that bullying may bring about hallucinatory experiences by placing a person at risk of developing intrusive cognitions. This possibility is suggested by research that has shown that bullied adolescents experience intrusive cognitions, similar to those seen in post-traumatic stress disorder (Idsoe, Dyregrov, & Idsoe, 2012). Given the findings

reported in Study 4, where participants who reported higher levels of intrusive thoughts also reported more hallucinatory experiences, it seems plausible that the association between bullying and AH could be mediated by intrusive cognitions. Research that examines whether this is the case is required.

Another avenue for future research is to examine the effect of negative affect on other tasks that are thought to be related to reality discrimination. For example, in Brookwell et al.'s (2013) meta-analysis of the association between reality discrimination performance and AH-proneness, the magnitude of the association between AH-proneness and source memory performance was also examined. In typical source memory tasks (e.g., Brébion et al., 2000), a participant and an experimenter perform a task where they must generate a set of items in response to a cue (e.g., "Name a type of fruit"). Later, the participant is presented with all of the items that were generated, and is asked to recall whether they or the experimenter generated each item. Consistent with models that propose that AH arise from an externalizing bias, AH-prone participants make more external misattributions (i.e., they identify self-generated items as having been generated by the experimenter) than do controls. Given that the association between AH-proneness and performance on source memory tasks was similar to the association between AH-proneness and performance on reality discrimination tasks, Brookwell et al. suggested that these two tasks measure the same process. One way in which to further investigate if this is the case would be to examine the effect of negative affect on source memory performance. Presumably, if reality discrimination and source memory tasks assess the same process, performance on both should be modulated by the same variables. Inducing negative affect should therefore lead participants to make more external misattributions on the source memory task, in the same way that it led to an increase in the number of external misattributions participants made on the reality discrimination task.

At present, two studies have examined the impact of an unpleasant stressor, which aimed to elicit psychobiological stress responses (e.g., an increase in salivary cortisol), on source memory performance (Smeets et al., 2006, 2008). However, in contrast to what would have been predicted from the findings of Study 5, both studies found that exposure to an unpleasant stressor reduced the number of external misattributions participants made. Two aspects of the design of Smeets and colleagues' studies, however, may have led to their results opposing what one would have predicted based on the findings of Study 5. First, both studies exposed participants to the stressor after the generation of items. The impact of the stressor will therefore have been upon recall of source information, rather than encoding of source information, and it is the latter that appears to be atypical in people who experience AH (Daprtati, Nico, Franck, & Sirigu, 2003; Sugimori, Asai, & Tanno, 2013). In addition, these studies employed either the Trier Social Stress Test (Kirschbaum, Perke, & Hellhammer, 1993) or the Cold Pressor Test (Lovallo, 1975) to induce stress responses in participants. These inductions require participants to perform a public speaking task and to submerge their hands in ice-cold water for up to three minutes, respectively. Clearly, these are very different to the induction employed in Study 5, which required participants to recall and write about an unpleasant autobiographical memory. These two factors may account for Smeets and colleagues' results being discordant with what one would have predicted given the findings of Study 5. Research that examines how the induction of negative affect, using the same method employed in Study 5, prior to the generation of a set of items modulates recall of the source of those items is required.

The findings presented in this thesis also indicate a number of potential avenues for novel therapeutic approaches. Very broadly, one could argue that Study 1's findings—that the association between low levels of maternal care and PLEs were mediated by exposure to bullying—support the use of interventions that improve levels of parental care and of

interventions that reduce the frequency of bullying of children and adolescents. Effective interventions to improve parental caregiving (Eshel, Daelmans, Cabral de Mello, & Martines, 2006) and to reduce bullying in schools (Ferguson et al., 2007; Ttofi & Farrington, 2011) have been developed. However, the effects of these interventions on maternal responsiveness and prevalence of bullying, respectively, appear to be small. More problematically, population-wide, psychosocial interventions strategies of this type are viewed sceptically (Kirkbride & Jones, 2011) and are unlikely to be implemented.

The findings of Study 4 and Study 5—that reality discrimination biases moderate the association between intrusive thoughts and AH-proneness and that reality discrimination biases can be modulated by mood—suggest a number of treatment options for AH. One option is to attempt to reduce the frequency of the occurrence of the raw material of AH. For example, if the triggers of intrusive thoughts can be identified, then therapy could involve learning ways to avoid these triggers, thus reducing the frequency with which a person experiences the ‘raw material’ of AH. Approaches such as acceptance and commitment-based therapy (Bach & Hayes, 2002) aim to reduce thought suppression, an approach which may reduce the number of ‘ironic rebounds’ a person experiences (Wegner & Zanakos, 1994), and so may reduce the number of intrusive cognitions a person experiences. A second option is to attempt to improve the reality discrimination abilities of people who experience AH. This may be the most practical option of the two, in that it would address a mechanism that may underlie multiple different types of AH, and because it may prove difficult to identify the triggers of intrusive thoughts (Kvavilashvili & Mandler, 2004).

While there is a small amount of evidence to suggest that anti-psychotic medication may improve a person’s reality discrimination abilities (e.g., Keefe et al., 2003), there are a number of other ways in which reality discrimination abilities could be improved. One possibility is that cognitive training could lead to improved reality discrimination skills. This

seems plausible, as a recent paper has shown that training on a battery of cognitive tasks—such as making fine-grain judgements under high working memory load and emotion recognition—improved the reality monitoring abilities of a group of patients with a diagnosis of psychosis (Subramaniam et al., 2012). This study, however, assessed reality monitoring using a source memory paradigm (i.e., participants had to recall whether they or the experimenter had generated a response), so it remains unclear whether cognitive training would have any impact on reality discrimination. In addition, Subramaniam et al. reported that the association between change in reality monitoring performance and change in psychotic symptomatology was not significant, although this may have simply been a result of low statistical power, as this association was moderate ($r = 0.27$). Research that examines whether cognitive training can modulate reality discrimination, rather than reality monitoring, would be of interest. If modulation of reality discrimination can be achieved through cognitive training, adequately powered clinical research that examines whether any changes in reality discrimination correlate with changes in the severity of psychotic experiences reported by patients would also be useful.

Another possibility is that neurostimulation, such as transcranial direct current stimulation (tDCS), of certain brain regions could be used to modulate reality discrimination. tDCS can be used either to increase (via anodal stimulation) or decrease (via cathodal stimulation) the excitability of cortical regions (Nitsche & Paulus, 2000) and two studies have shown that cathodal stimulation of left temporo-parietal cortex reduces the frequency of AH in psychotic patients (Brunelin et al., 2012; Homan et al., 2011). Moseley, Fernyhough, and Ellison (in press) have argued that these findings may be explained in terms of modulation of this region leading to changes in reality discrimination biases, which reduce the likelihood of the occurrence of hallucinatory experiences. Consistent with this argument, one recently completed study has shown that participants make fewer external misattributions on a reality

discrimination task following cathodal stimulation of the left temporo-parietal junction, in comparison to anodal stimulation of this same region (Moseley, Fernyhough, & Ellison, in preparation). Further examination of the possibility that tDCS can modulate reality discrimination in such a way as to reduce AH would be helpful.

Finally, the finding reported in Study 5—that negative affect can elicit biased reality discrimination—supports arguments (e.g., van der Meer, van't Wout, & Aleman, 2009) that therapeutic approaches that attempt to improve emotion regulation (e.g., Greenberg & Bolger, 2001) may be of benefit to people with psychosis. The results presented here suggest that improving a person's emotion regulation abilities will not only enable them to cope with psychotic experiences, but would also help prevent the onset of psychotic experiences by, for example, reducing negative affect, and hence protecting a person from negative affect-induced biases in reality discrimination. This claim is consistent with current therapeutic approaches for persecutory delusions that place less emphasis on attempting to modify delusional beliefs, and place a greater emphasis on reducing negative emotion processes, such as worry (e.g., Foster, Startup, Potts, & Freeman, 2010; Freeman, Dunn, Startup, & Kingdon, 2012). These interventions may be preferable to traditional therapeutic approaches, as they attempt to ameliorate the affective problems that appear to underlie at least some psychotic experiences, and because they focus on problems that patients identify as issues that have a negative impact on their quality of life (Freeman et al., 2012).

7.4 Limitations

As has been noted elsewhere in this Discussion, one limitation of the studies reported in this thesis is that all employed non-clinical participants, and it will be important to replicate these studies with clinical samples. In addition, two of the studies reported here (Study 1 and Study 2) employed mediation analysis, and so it is worthwhile to note two important limitations of this approach. The first is that an unmeasured confounder could

account for the mediation effects reported (Cole & Hernán, 2002). That is, some unmeasured variable may be associated with both the mediator and the outcome variable, and the apparent mediating effect is simply a result of those associations. There is little that can be done to address this concern, other than to control for as many potential confounders as is practical and to acknowledge the possibility that these results may have been affected by an unmeasured confounding variable (Cole & Hernán, 2002). The second is that mediation analysis is not able to demonstrate causal relations between variables (Mackinnon, Fairchild, & Fritz, 2007). For example, in Study 2, given the results of the mediation analysis, it is fair to state that the association between attachment anxiety and paranoid thinking can, in part, be accounted for by the associations between attachment anxiety and loneliness and between loneliness and paranoid thinking. However, the mediation analysis does not provide evidence that there are causal relations between these variables. As has been recommended elsewhere in this Discussion, longitudinal research would be helpful in establishing the causal relations between these variables.

7.5 Summary

The five studies reported in this thesis have demonstrated a number of ways in which social, cognitive, and emotional variables are associated with psychotic experiences. Importantly, these studies suggest that these factors do not operate independently of one another. Rather, these factors appear to mediate and moderate one another in complex ways to foster, or to preclude, the development of psychotic experiences, at least in non-clinical samples. A number of avenues for future research have been outlined in this General Discussion, which would address several outstanding research questions, including questions that will integrate neurobiological factors with the social, cognitive, and emotional factors considered here.

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